MILITARY MEDICAL MANUALS

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SIR ALFRED KEOGH, G.C.B., M.D., F.R.C.P.
LT-GEN. T. H. J. C. GOODWIN, C.B., C.M.G., D.S.O.

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ELECTRO-DIAGNOSIS IN WAR

ZIMMERN & PEROL

EDITED BY
E. P. CUMBERBATCH

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ELECTRO-DIAGNOSIS IN WAR

CLINICAL. MEDICAL BOARD TECHNIQUE AND INTERPRETATION

By

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GENERAL INTRODUCTION

The infinite variety of injuries which any war presents to the surgeon gives to military surgery a special interest and importance. The special interest and importance, in a surgical sense, of the great European War lies not so much in the fact that examples of every form of gross lesion of organs and limbs have been seen, for if we read the older writers we find little in the moderns that is new in this respect, but is to be found in the enormous mass of clinical material which has been presented to us and in the production of evidence sufficient to eliminate sources of error in determining important conclusions. For the first time also in any campaign the labours of the surgeon and the physician have had the aid of the bacteriologist, the pathologist, the physiologist, and indeed of every form of scientific assistance, in the solution of their respective problems. The clinician entered upon the great war armed with all the resources which the advances of fifty years had made available. If the surgical problems of modern war can be said not to differ sensibly from the campaigns of the past, the form in which they have been presented is certainly as different as are the methods of their solution. The achievements in the field of discovery of the chemist, the physicist and the biologist have given the military surgeon an advantage in diagnosis and treatment which was denied to his predecessors, and we are able to measure the effects of these advantages when we come to appraise the results which have been attained.

But although we may admit the general truth of these statements, it would be wrong to assume that modern scientific knowledge was, on the outbreak of the war, immediately useful to those to whom the wounded were to be confided. Fixed principles existed in all the sciences auxiliary to the work of the surgeon, but our scientific resources were not immediately available at the outset of the great campaign; scientific work bearing on wound problems had not been arranged in a manner adapted to the requirements—indeed, the requirements were not fully foreseen; the workers in the various fields were isolated, or isolated themselves, pursuing new researches rather than concentrating their powerful forces upon the one great quest.

However brilliant the triumphs of surgery may be—and that they have been of surpassing splendour no one will be found to deny—experiences of the war have already produced a mass of facts sufficient to suggest the complete remodelling of our methods of education and research.

The series of manuals, which it is my pleasant duty to introduce to English readers, consists of translations of the principal volumes of the "Horizon" Collection, which has been appropriately named after the uniform of the French soldier.

The authors, who are all well-known specialists in the subjects which they represent, have given a concise but eminently readable account of the recent acquisitions to the medicine and surgery of war which had hitherto been disseminated in periodical literature.

No higher praise can be given to the Editors than to say that the clearness of exposition characteristic of the French original has not been lost in the rendering into English.

MEDICAL SERIES

The medical volumes which have been translated for this series may be divided into two main groups, the first dealing with certain epidemic diseases, including syphilis, which are most liable to attack soldiers, and the second with various aspects of the neurology of war. The last word on *Typhoid Fever*, hitherto "the greatest scourge of armies in time of war," as it has been truly called, will be found in the monograph by MM. Vincent and Muratet, which contains a full account of recent progress in bacteriology and epidemiology as well as the clinical features of typhoid and paratyphoid fevers. The writers combat a belief in the comparatively harmless nature of paratyphoid and state that in the present war hæmorrhage and perforation have been as frequent in paratyphoid, as in typhoid fever. In their chapter on diagnosis they show that the serum test is of no value in the case of those who have undergone anti-typhoid or anti-paratyphoid vaccination, and that precise information can be gained by blood cultures only. The relative advantages of a restricted and liberal diet are discussed in the chapter on treatment, which also contains a description of serum-therapy and vaccine-therapy and the general management of the patient.

Considerable space is devoted to the important question of the carrier of infection. A special chapter is devoted to the prophylaxis of typhoid fever in the army. The work concludes with a chapter on preventive inoculation, in which its value is conclusively proved by the statistics of all countries in which it has been employed.

MM. Vincent and Muratet have also contributed to the series a work on *Dysentery, Cholera and Typhus* which will be of special interest to those whose duties take them to the Eastern Mediterranean or Mesopotamia. The carrier problem in relation to dysentery and cholera is fully discussed, and special stress is laid on the epidemiological importance of mild or abortive cases of these two diseases.

In their monograph on *The Abnormal Forms of Tetanus*, MM. Courtois-Suffit and Giroux treat of those varieties of the disease in which the spasm is confined to a limited group of muscles, *e.g.* those of the head, or one or more limbs, or of the abdomino-thoracic muscles. The constitutional symptoms are less severe than in the generalised form of the disease, and the prognosis is more favourable.

The volume by Dr. G. Thibierge on *Syphilis and the Army* is intended as a *vade mecum* for medical officers in the army.

Turning now to the works of neurological interest, we have two volumes dealing with lesions of the peripheral nerves by Mme. Athanassio-Benisty, who has been for several years assistant to Professor Pierre Marie at La Salpêtrière. The first volume contains an account of the anatomy and physiology of the peripheral nerves, together with the symptomatology of their lesions. The second volume is devoted to the prognosis and treatment of nerve lesions.

The monograph of MM. Babinski and Froment on *Hysteria or Pithiatism and Nervous Disorders of a Reflex Character* next claims attention. In the first part the old conception of hysteria, especially as it was built up by Charcot, is set forth, and is followed by a description of the modern conception of hysteria due to Babinski, who has suggested the substitution of the term "Pithiatism," *i.e.* a state curable by persuasion, for the old name hysteria. The second part deals with nervous disorders of a reflex character, consisting of contractures or paralysis following traumatism, which are frequently found in the neurology of war, and a variety of minor symptoms, such as muscular atrophy, exaggeration of the tendon reflexes, vasomotor, thermal and secretory changes, etc. An important section discusses the future of such men, especially as regards their disposal by medical boards.

An instructive companion volume to the above is to be found in the monograph of MM. Roussy and Lhermitte, which embodies a description of the psychoneuroses met with in war, starting with elementary motor disorders and concluding with the most complex represented by pure psychoses.

SURGICAL SERIES

When the present war began, surgeons, under the influence of the immortal work of Lister, had for more than a quarter of a century concerned themselves almost exclusively with elaborations of technique designed to shorten the time occupied in or to improve the results obtained by the many complex operations that the genius of Lister had rendered possible. The good behaviour of the wound was taken for granted whenever it was made, as it nearly always was, through unbroken skin, and hence the study of the treatment of wounds had become largely restricted to the study of the aseptic variety. Septic wounds were rarely seen, and antiseptic surgery had been almost forgotten. Very few of those who were called upon to treat the wounded in the early autumn of 1914 were familiar with the treatment of grossly septic compound fractures and wounded joints, and none had any wide experience. To these men the conditions of the wounds came as a sinister and disheartening revelation. They were suddenly confronted with a state of affairs, as far as the physical conditions in the wounds were concerned, for which it was necessary to go back a hundred years or more to find a parallel.

Hence the early period of the war was one of earnest search after the correct principles that should be applied to the removal of the unusual difficulties with which surgeons and physicians were faced. It was necessary to discover where and why the treatment that sufficed for affections among the civil population failed when it was applied to military casualties, and then to originate adequate measures for the relief of the latter. For many reasons this was a slow and laborious process, in spite of the multitude of workers and the wealth of scientific resources at their disposal. The ruthlessness of war must necessarily hamper the work of the medical scientist in almost every direction except in that of providing him with an abundance of material upon which to work. It limits the opportunity for deliberate critical observation and comparison that is so essential to the formation of an accurate estimation of values; it often compels work to be done under such high pressure and such unfavourable conditions that it becomes of little value for educative purposes. In all the armies, and on all the fronts, the pressure caused by the unprecedented number of casualties has necessitated rapid evacuation from the front along lines of communication, often of enormous length, and this means the transfer of cases through many hands, with its consequent division of responsibility, loss of continuity of treatment, and absence of prolonged observation by any one individual.

In addition to all this, it must be remembered that in this war the early conditions at the front were so uncertain that it was impossible to establish there the completely equipped scientific institutions for the treatment of the wounded that are now available under more assured circumstances, and that progress was thereby much hampered until definitive treatment could be undertaken at the early stage that is now possible.

But order has been steadily evolved out of chaos, and many things are now being done at the front that would have been deemed impossible not many months ago. As general principles of treatment are established it is found practicable to give effect to them to their full logical extent, and though there are still many obscure points to be elucidated and many methods in use that still call for improvements, it is now safe to say that the position of the art of military medicine and surgery stands upon a sound foundation, and that its future may be regarded with confidence and sanguine expectation.

The views of great authorities who derive their knowledge from extensive first-hand practical experience gained in the field cannot fail to serve as a most valuable asset to the less experienced, and must do much to enable them to derive the utmost value from the experience which will, in time, be theirs. The series covers the whole field of war surgery and medicine, and its predominating note is the exhaustive, practical and up-to-date manner in which it is handled. It is marked throughout not only by a wealth of detail, but by clearness of view and logical sequence of thought. Its study will convince the reader that, great as have been the advances in all departments in the services during this war, the progress made in the medical branch may fairly challenge comparison with that in any other, and that not the least among the services rendered by our great ally, France, to the common cause is this brilliant contribution to our professional knowledge.

A glance at the list of surgical works in the series will show how completely the ground has been covered. Appropriately enough, the series opens with the volume on *The Treatment of Infected Wounds*, by A. Carrel and G. Dehelly. This is a direct product of the war which, in the opinion of many, bids fair to become epoch-making in the treatment of septic wounds. It is peculiar to the war and derived directly from it, and the work upon which it is based is as fine an example of correlated work on the part of the chemist, the bacteriologist and the clinician as could well be wished for. This volume will show many for the first time what a precise and scientific method the "Carrel treatment" really is.

The two volumes by Professor Leriche on *Fractures* contain the practical application of the views of the great

Lyons school of surgeons with regard to the treatment of injuries of bones and joints. Supported as they are by an appeal to an abundant clinical experience, they cannot fail to interest English surgeons, and to prove of the greatest value. It is only necessary to say that *Wounds of the Abdomen* are dealt with by Dr. Abadie, *Wounds of the Vessels* by Professor Sencert, *Wounds of the Skull and Brain* by MM. Chatelin and De Martel, and *Localisation and Extraction of Projectiles* by Professor Ombrédanne and R. Ledoux-Lebard, to prove that the subjects have been allotted to very able and experienced exponents.

ALFRED KEOGH.

PREFACE

The subject of the present volume is the use of electrical methods in the diagnosis and prognosis of the injuries and diseases of the nervous system which are encountered in war. The first two chapters deal with the electrical reactions of muscles and nerves in cases of paralysis, the methods of obtaining them, the different types of reaction, and the light which they throw on the site and nature of the lesion which may be present.

It is of the utmost importance that every method of investigation should be utilised which can throw light on the lesion in nerve injury, because the nature of the latter will determine the treatment to be adopted and the subsequent usefulness of the patient, either for continuation of military service or for employment in civil life.

The value of electrical testing has always been admitted in cases of *disease* of the nervous system, as the discovery of the reaction of degeneration is irrefutable evidence of the situation of the lesion in the lower motor neurone. In the case of nerve *injury*, the problem is different and more difficult, because the information desired is not merely the situation of the lesion, but its severity and the treatment which should be given, whether surgical or physiotherapeutic. It was hoped that electrical testing of the reactions would provide the necessary information. Its failure to do so in many cases is responsible for the scepticism which prevails in some quarters regarding the value of electrical testing. The manner in which the test is frequently carried out, the use of unstandardised coils and unmeasured stimuli, the use of the faradic current only and the performance of only one test, cannot yield the information which thorough and repeated testing can provide.

A study of the present work will show the value of a careful and complete investigation of the electrical reactions. The authors describe the method which they have found most trustworthy, and they emphasise the importance of making, not one, but several tests, repeated at intervals, because the abnormal reactions of muscle and nerve are not permanent but varying reactions, and their changes from week to week, or month to month, afford information which cannot be obtained by a single test.

The importance of investigating other physical signs as well as the electrical reactions is recognised; and while the authors make no undue claim for the latter, they mention how cases of serious nerve injury which had not been recognised by clinical examination, were afterwards made manifest by electrical testing. One of the great advantages of electrical testing is that it affords definite and indisputable objective signs in which the personal element of the patient plays no part.

In Chapter IV is described an electrical method of testing cutaneous sensation, its advantages being that the stimulus is measured and can be repeated on subsequent occasions.

A galvano-psychic reaction is described. It appears to be a valuable test in deciding whether loss of function of sensory nerves or nerves of special sense is simulated or due to organic disease.

Chapter V contains a lucid account of the various reactions which follow the application of the galvanic current to the head so that the internal ear is traversed by the current. These reactions, viz. nystagmus, sensation of vertigo, and angular deviation from the straight line when walking, are due to stimulation of the vestibule and semicircular canals. The test is of value when applied to cases presenting cerebral symptoms. The presence of abnormal reactions indicates that the symptoms are due to injury or organic disease and are not psychical or simulated.

Short accounts are given of the use of condensers in testing, the chronaxie and its measurement, and of the theories regarding the various reactions. Good illustrations are given of the position of the motor points, and their situation is described, together with the action of the various muscles. These additions render the present work the most complete account that has yet appeared of the uses of electrical methods in the investigation of paralysis occurring in war.

E. P. C.

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INTRODUCTION

The importance of electro-diagnosis in cases of gunshot wounds, and a desire to afford precise and useful information to neurologists, physiotherapists, surgeons, and members of Medical Boards, has led to our making certain notes on the essential principles of electrical investigation. In arranging these, the present work has been produced, which we trust will prove of service to our colleagues.

Electro-diagnosis is an indispensable accessory to clinical examination in war neurology. Systematically carried out, it is of the greatest assistance in solving a whole series of problems arising in daily practice. Its value lies in the fact that electrical reactions are definite, indisputable, and absolutely precise objective signs.

It is this method which, for instance, obviates indecision when the neurologist suspects the presence or co-existence of any form of hysteria. It provides the fundamental data for prognosis in nerve injuries, and thus affords definite indications for treatment. It also serves to demonstrate the degree of any loss of function before a Medical Board.

Unfortunately, for various reasons, too much has been expected of electro-diagnosis; questions have been referred to it which it cannot decide, and but for the proof to which it has been put since the days of Duchenne (of Boulogne) and Erb, these demands on the method might well have resulted in its partial discredit.

A widespread belief still exists that the results of electrical investigation should, in some sense, automatically determine the capability of paralysed nerves and muscles to perform their functions. This is a misconception. In the first place this capability cannot be expressed in equivalents, and in the second, it should not be forgotten that the electrical excitability of the neuro-muscular apparatus is a property entirely different and distinct from its ability to initiate movement following stimulation of the higher centres. Excitability is not a criterion of the state of the nerve; it simply gives a valuable indication of its condition.

As with other laboratory methods, electro-diagnosis is utilised clinically to decide or confirm a differential diagnosis. The reaction of degeneration shows the presence of a peripheral motor neurone lesion as certainly as the agglutination reaction demonstrates a typhoid infection.

But the range of electrical investigation is not limited to verifying the presence or absence of the reaction of degeneration. The degrees of abnormality in electric reactions, the way in which they are associated, and their development, are so many facts an analysis of which will show the nature of the case concerned, determine the prognosis, and indicate the form of treatment.

If such an investigation is to be complete and productive of results, it is obvious that it must always be exact and painstaking, and occupy a considerable length of time. As a rule no conclusions can be drawn immediately, first because it will often be necessary to postpone the repetition of an important test which has given doubtful results, or no results, at the first examination, and also because, in the vast majority of cases, the information afforded by subsequent development of the reactions is indispensable. The case must be followed up step by step from the electro-diagnostic point of view, as is usual from the clinical. With electrical reactions—and we cannot too strongly emphasise this point—it is as with the temperature. A single rise of temperature does not give the picture of pyrexia; it is the chart that should be considered.

It follows obviously from what has been said that where examinations, often of extreme delicacy, are concerned, it is desirable first, that electrologists should agree to adopt a standard technique, and in the second place, that an individual case should always be examined by the same operator, when, as is usually the case, successive examinations are necessary. This ideal is perhaps less difficult to realise than the exigences of evacuating wounded would appear to suggest.

The duty of the medical electrician is not merely to note the particulars of abnormal reactions. When these have been determined, he must correlate them with the clinical symptoms, taking a whole series of facts into account: the probable nature of the injury or nerve lesion, the peculiarities of the nerve involved, the length of time which has elapsed since signs of a lesion were first observed, etc.

The duty of the surgeon and the neurologist is to translate the results of the investigation into clinical language.

It will therefore be evident that the practice of electro-diagnosis requires, not merely that the physician should be familiar with the difficulties of electrical technique, but also that he should have extensive neurological experience, founded on a thorough knowledge of anatomy.

Failing this, he will be unable to interpret the results of his investigation satisfactorily, or to form correct conclusions.

The value of electro-diagnosis depends on the physician who practises it.

ELECTRO-DIAGNOSIS IN WAR

CHAPTER I

THE TECHNIQUE OF ELECTRO-DIAGNOSIS

I. DEFINITION

ELECTRO-DIAGNOSIS may be defined as the summation of the results which can assist the clinician by determining the mode of reaction of the organism to different forms of electrical current.

The term more particularly suggests a process of exploration, based on the responses of motor nerves and muscles to electrical stimulation. Making an electro-diagnosis signifies, as a matter of fact, an investigation made to ascertain whether, in a patient suffering from some affection of the neuro-muscular system, the electrical reactions of the muscles and motor nerves differ from the normal physiological reactions, and if so, an examination of the nature of the difference.

It is not, however, merely for motor nerves that electrical investigation can afford evidence of clinical value. Sensory nerves may also, in affections which involve them directly or indirectly, present alterations of excitability, into which it will sometimes be of interest to inquire. Similarly with certain reflex phenomena, among which is that known as voltaic vertigo, anomalies of which may reveal even slight disturbances of the mechanisms which govern them.

II. APPARATUS REQUIRED

The apparatus necessary for electro-diagnosis comprises :

1. A faradic apparatus.
2. A galvanic apparatus.
3. Electrodes for application of currents, and conducting wires.

Some electrologists prefer to use condensers of different capacities in the place of galvanic and faradic apparatus.

Faradic Apparatus.—We shall presuppose a knowledge of the theory and structure of medical induction coils, with their primary and secondary windings, their interrupters, condensers, etc.

The commonest are sledge coils (fig. 1), in which the secondary can be made to approach and cover the primary coil to any desired extent. Some forms of this apparatus are intended to remain fixed on the operator's table, others are portable. They are supplied from accumulators, two or three Leclanché cells, or two dry cells.

In order to standardise the results of electrical examination, it is desirable that all faradic apparatus should be supplied with a current of *fixed voltage* (4 volts, for instance). Similarly, there should be an agreement between instrument-makers whereby the *resistance* (measured in ohms) of the two secondary coils which they manufacture (coarse and fine winding) should be *constant*.[1] It is useful to have an interrupter, with which a tetanising current can be produced, as well as a single-wave current. One of the most useful types of apparatus from this point of view is the lever interrupter, which will give any frequency of interruption from 50 to 3,000 per minute.

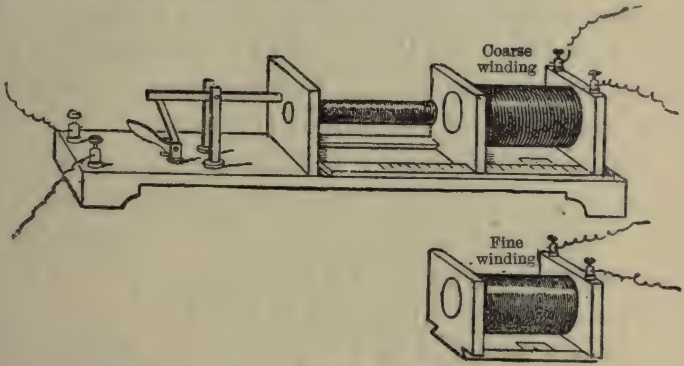

FIG. 1.—Faradic sledge coil.

The intensity of the faradic current is increased by approximating the secondary and primary coils, and for clinical purposes it is considered sufficient to measure this intensity in terms of the distance (in centimetres) between the two coils.[1]

---

[1] The secondary coils of the present instruments have a resistance of 1 to 10 ohms in the coil with coarse wire, and 500 to 1,000 ohms in the coil with fine wire.

Galvanic Apparatus.—If the continuous current of an electric-light circuit is to be utilised, a *reducer of potential* will afford a suitable current at its distal terminals. A *rheostat* is sometimes used, instead of a potential-reducer, to graduate the current.

Rheostats are of the solid or "liquid" type. They are introduced into the circuit as an additional resistance, variable at will. As with the potential-reducer, the graduation of the current is smooth.

Urban electric-supply systems, producing electric energy by continuous current dynamos, in reality supply an undulatory current which is equivalent, for practical purposes, to a continuous current. The frequency of the undulation is usually so great and the waves so small that it produces no appreciable effect in the stimulation of nerves and muscles. At the same time it is always preferable to use a source of absolutely pure and unvarying continuous current. The best source, from this point of view, is a *battery of accumulators* (30 to 40 cells).

Since accumulators require attention and recharging, a *cell-battery* is sometimes preferred. These should be of sufficient capacity to ensure a satisfactory supply. Cells of the Leclanché type, using ammonium or zinc chloride, and without the porous pot, are the most popular.

---

[1] Instruments for measuring the faradic current in units of electricity have not yet been used in practice; this is to be regretted, since, with many varieties of apparatus, results obtained by different observers cannot be compared.

No forms of the above-described apparatus are portable. Hence manufacturers have produced *portable cell-batteries*, in the usual model of which mercury bisulphate is the liquid element (fig. 2). A current of sufficient intensity for all the present needs of electro-diagnosis is ensured with 30 to 40 cells.

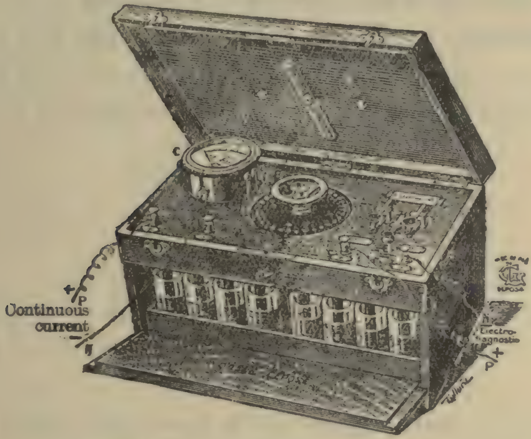

Fig. 2.—Portable galvanic battery.

Most portable batteries are fitted with an unsatisfactory appliance for graduation, the collector. A collecting arm, by passing over a series of "buttons," introduces the cells successively into the circuit. Shifting from one button to the next causes a sudden rise in E.M.F. equivalent to the voltage of the corresponding cell; this is a disadvantage in investigating quantitative changes of excitability, one, however, which may easily be corrected by the introduction of a rheostat.

The intensity of a continuous current is measured by means of the milliamperemeter. This, to be read easily, should be *aperiodic*; that is to say, the indicating needle should come immediately to rest in the correct position without any periodic oscillation.

Permanent and Varying States. Simple and Double Key.—In the continuous current, the *permanent* state and the *varying states* are distinguished. The current is said to pass into a permanent state when its intensity does not vary as time passes. By varying states during "make" and "break," we understand the periods while the current is being made and being broken, and, more particularly, *rapid* make and *rapid* break (fig. 3).

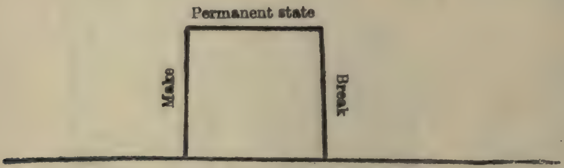

FIG. 3.—Permanent state of the constant current, and the varying states, at make and at break.

The varying states of make and break, known also as make and break waves, induce muscular contractions when the current intensity is sufficient. To produce these contractions in a convenient way, an interrupter, such as the Morse key (the key of telegraphic transmitters), also known as the "simple" key, is introduced into the circuit. When the key is depressed the circuit is suddenly made; when it is released it springs back, and the circuit is broken.

The interrupter is sometimes fixed on the handle of the exploring electrode (interrupter handle).

To enable the polarity of the electrodes to be reversed easily, a current reverser is introduced into the circuit, in front of the Morse key. The functions, however, of reverser and interrupter are excellently combined, from the practical point of view, in the "double key."

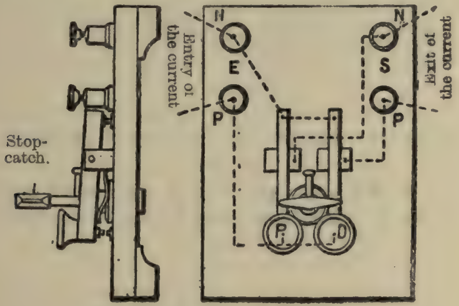

FIG. 4.—Courtade's key.

Courtade's "interrupter-reverser," or "Courtade's key" (fig. 4), consists of a double key, each extremity of which can make contact with two studs—anterior and posterior. The anterior studs represent the bifurcated extremity of one conducting wire from the battery, the posterior the bifurcated extremity of the wire from the other pole of the battery.

The wires to the electrodes are attached to the pivots of the keys.

The position of the keys in fig. 5 is that of rest. It will be seen that when both keys are *raised*, no current is conducted. But if the key A (fig. 6) is lowered, its posterior extremity breaks contact with the posterior stud, and its anterior makes contact with the anterior stud; in this way the circuit is made. By following out the wire connections in the diagram it will be seen that the negative pole of the battery is then in connection with the active electrode; this is therefore a *negative make* (fig. 6). When contact is broken at A, *i.e.* when the key A returns to the position of rest, the circuit is broken, and thus a *negative break* has been produced.

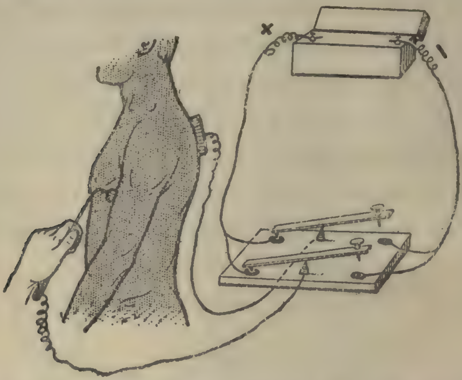

Fig. 5.—Manipulation of the double key (position of rest).

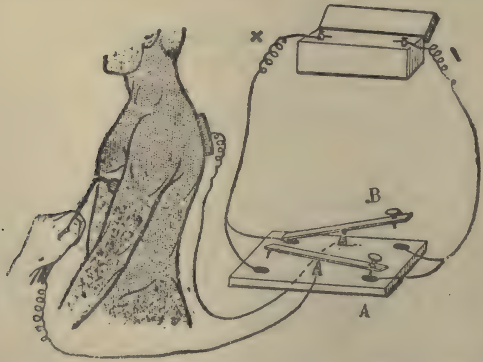

Fig. 6.—Manipulation of the double key. (The key A is lowered. In this position, the exploring electrode is connected to the negative pole.)

If the key B is now lowered, a make is again produced, but since the negative pole of the cell has thus been connected with the passive electrode, the active electrode will be *positive*.

By simply manipulating the keys, this instrument can therefore be used to transpose the polarity of the electrodes, and to effect make and break. Its convenience renders it almost indispensable in galvanic exploration.

In the portable galvanic battery which one of us has had constructed (fig. 2), the double key mechanism is fitted alongside the apparatus for graduation and measurement.

The organism reacts to the continuous electric current as an electrolyte, and consequently follows the laws of electrolytic polarisation. In other words, the passage of the electric current develops an electromotive force of polarisation within the organism, antagonistic to that of the inducing current. The intensity of this current may reach such a degree in certain conditions that the results of electrical exploration may be affected by it. It will readily be observed from the diagram of Courtade's key that at the moment of break (release of the key) (fig. 5), the polarisation current, induced throughout the make, is short-circuited across the key. This is an advantage from the point of view of the next make of the exploring current, but at the moment of break, the make of the polarisation current superimposes its effects on those of the break of the exploring current. Since the galvanometer registers the sum of the intensities of the two superimposed currents, it follows that, in determining the "break" threshold, the intensity recorded will not be the actual intensity required for the threshold stimulus.

The double key is therefore a defective method for studying breaks; preferable to it is a simple key which does not make the polarisation current at the break of the exploring current (Bourguignon).[1] On the other hand, the passage of the polarisation current through the double key in the interval between two makes of the exploring current obviates interference with the latter by any secondarily induced current. As it is usually considered sufficient in electro-diagnosis to investigate make contractions, it will be seen that with the double key these can always be obtained pure.

In permanent installations the necessary instruments for electro-diagnosis are usually mounted on a wall-bracket or a table. These *electro-diagnosis tables* are arranged in such a way that both faradic and galvanic current are connected with a single apparatus for utilising them, which renders unnecessary the disconnection of the wires. This commutation of the current is effected by a two-way switch, known as *Watteville's commutator*.

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[1] If one of the keys is kept permanently down, the Courtade key becomes a simple key.

Lastly, an *interrupting metronome* effects rhythmic interruptions and reversals of the current, acting, if desired, in place of manual manipulation of the Courtade key. Some more complicated electro-diagnosis tables are also fitted with various instruments, voltmeters, condensers, etc.

Electrodes.—In exploring motor nerves and muscles the current is applied by means of *sponge-electrodes*. Of these there are two forms, the *flat* and the *button-shaped*. The former (fig. 7) consist of a malleable metallic plate, unfortunately still sometimes supplied by the manufacturers covered with chamois leather, but which it is more satisfactory to cover with a layer of absorbent cotton material. Flat electrodes act as what are known in electro-diagnostic technique as the *indifferent* electrodes, since their only purpose is to make the circuit through the patient; and they

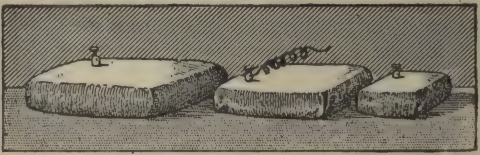

FIG. 7.—Flat electrodes.

can consequently be placed anywhere on the surface of the body. It is usual to apply them to the middle of the chest or back, where they are secured by means of an elastic band, after being moistened with warm water.

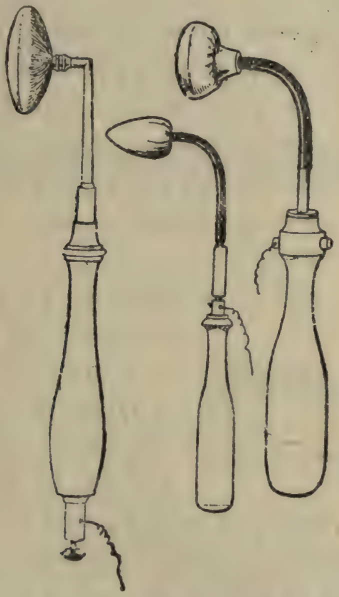

FIG. 8.—Exploring electrodes.

Button-shaped electrodes, known also as tampons, (fig. 8), consist of a carbon extremity covered with chamois leather or absorbent material, attached to the end of a straight or curved handle.

Used to localise the stimuli to the nerve or muscle, they constitute, in electro-diagnosis, the *active electrode*, the testing, or differentiating electrode.

The dimensions of the exploring electrode should always depend on those of the organ concerned. In the case of large muscles, electrodes of large diameter (2 to 3 cm.) are used; for small muscles, electrodes of smaller surface (5 to 8 mm. in diameter), or olive-shaped electrodes.

The exploring electrode introduces into the organism a current of the same intensity as does the indifferent electrode, but through a much smaller surface of entry. The relation between the intensity and the surface area is known as the *density* of the current. The density therefore increases with a decrease in size of the electrode.

In certain cases, particularly with the faradic current, it is necessary to localise the current to a single nerve or muscle. To apply this "bipolar" method it is best to use two small olive-shaped tampons held in one hand.

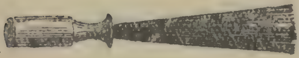

FIG. 9.—Duchenne's brush.

An investigation of cutaneous sensations with the faradic current is sometimes carried out with sponge electrodes, but it is more usual to employ metal electrodes, such as *Duchenne's brush* (fig. 9), a small brush formed by a bunch of brass wires.

Duchenne's brush can only be used for unipolar exploration. Now in cases of complete anæsthesia or marked hypoæsthesia, the strong currents which will have to be employed will affect both nerves and muscles simultaneously, the more so as the exploration is continued nearer to motor points. Contractions produced in this way will interfere considerably with the investigation of sensation. It is therefore well to adopt the bipolar method, which can be done by employing some bipolar metallic electrode, connected by two wires with the coil. *Tripier's rake* (fig. 10) is constructed on this principle: a racquet-shaped instrument from which project two sets of blunt brass points, respectively connected with the two poles of the coil.

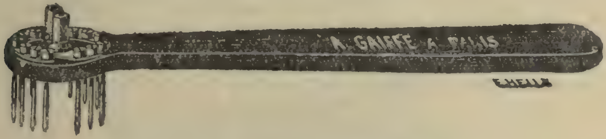

FIG. 10.—Tripier's rake.

Condensers.—The use of the condenser in electro-diagnosis has been vigorously championed in Austria by Zanietowski, and in France by Doumer and Cluzet. Considered too complex, however, owing to the calculations which it involves, it has been almost systematically discarded by the majority of electrotherapists. It is not, however, impossible that a reorganisation of the system of electro-diagnosis may reinstate it as a generally accepted method.

For reasons which will be explained later, certain authors have proposed to employ the method of condensers, *charged to varying degrees*, in electro-diagnosis. In this method condensers of different capacity are charged to an increasing voltage, thereby determining for any given condenser the smallest voltage which will evoke the threshold of muscular contraction.

Instead, however, of determining the minimal stimulus by varying the potential of the discharge from a standard condenser, it may be determined by means of a constant potential and a series of condensers of increasing capacity. This is the principle of the "method of condensers of variable capacity," the use of which, though still limited, may be required more generally in the future.

The duration of the discharge from a condenser is in proportion to its capacity.[1] With a series of condensers of increasing capacities, and charged to the same potential, it will therefore be possible to apply discharges to the nerve or muscle differing in the matter of *duration.*

Cluzet's *condenser of variable capacity* (fig. 11) consists of a series of condenser plates of respective capacities 0·005, 0·01, 0·02, 0·02, 0·05, 0·1, 0·2, 0·2, 0·5, 1, 3, and 5 microfarads.

On the upper surface of the box in which they are enclosed is a system of spring keys by means of which a single condenser, or a combination of any number, may be used. Capacities graduated from 0·005 to 10 microfarads are therefore available.

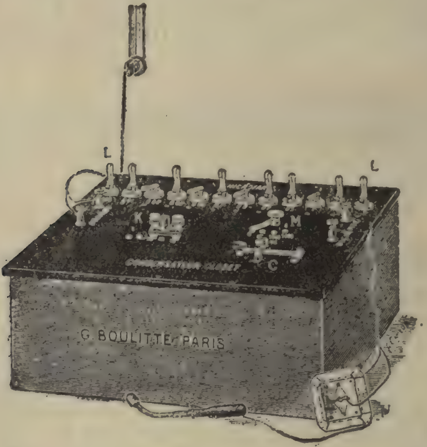

FIG. 11.—Condenser of variable capacity.

A plug connection supplies the apparatus from a source of continuous current (preferably a 110 volt circuit). A two-way Morse key effects successive charge and discharge into the utilisation circuit. Finally, a series of resistances (1,000, 2,000, 5,000 ohms) may also be introduced into this circuit. The current is conveyed to the patient by means of ordinary electrodes (active and indifferent); the polarity is reversible at will by a commutator. No measuring instrument is fitted; this, however, would serve no purpose, since the determination of the capacity which gives the threshold (liminal capacity), decides the principal question of the investigation, the duration of stimulus. For normal muscles and nerves this liminal capacity is almost exactly 0·02 microfarad. But in a pathological condition this is inadequate. By manipulating the keys higher capacities are gradually introduced until the minimal contraction is obtained.

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[1] To be more exact, the duration of the discharge from a condenser depends upon the capacity C and the resistance R. The product $RC = t$ is the time constant of the condenser. If R is constant, the duration of the discharge varies solely with the capacity of the condenser.

Sometimes, particularly when the muscle reacts to the galvanic current with a very slow contraction, a capacity of 10 microfarads produces no contraction. One of the resistances must then be introduced into the discharge circuit.

III. THE NORMAL REACTION OF MOTOR NERVES AND MUSCLES

The Meaning of Threshold.—All modes of electrical stimulation, such as the faradic current, static discharges, discharges from condensers, and varying states of make and break of the galvanic current, if applied to a healthy muscle or its motor nerve, will produce muscular contraction; that is to say, the passage of the muscle from the state of repose into that of activity.

For the effects of stimulation to be produced, the stimulating agent must reach a certain definite measure, below which it remains ineffective. This lower limit is the threshold stimulus, and the contraction which it evokes, in response, is the minimal contraction.

All stronger stimuli cause contraction, and the energy of the latter increases with the intensity of the exciting current. This proceeds until a limit is reached, known as the maximal contraction, the energy of which cannot be increased by an increase of current-intensity. The point at which this maximum is reached is difficult to determine, but the value of the exciting current, at the moment of transition from an inadequate to an effective stimulus, is capable of much easier and more exact measurement. For this reason the threshold has been adopted in practice as a measure of excitability.

Excitability and Conductivity.—A motor nerve causes a muscle to contract only when the stimulus is conducted to the extremity of that nerve. This property of the nerve by which the stimulus is conducted along it, conductivity, is absolutely distinct from excitability. Several experiments in elementary physiology demonstrate the fact that these two properties of the nerve are independent. Of these the most classical is Grunhagen's experiment.

A nerve-muscle preparation is obtained, and the nerve is passed into a tube where a current of carbonic acid is circulating. It can then be shown that if the nerve be stimulated in its course through the tube, there is no muscular response. On the other hand, stimulation further up the nerve produces muscular contraction; conductivity is therefore preserved. The opposite phenomenon, however, is produced when the nerve is submitted to the action, not of acid, but of alcohol or ether vapour. In this case the conductivity is the first to disappear.

The independence of excitability and conductivity is similarly observed in electro-diagnosis.

Thus, Erb was the first to observe that in the common radial paralysis caused by momentary compression, stimulation of the nerve above the point of compression (in the axilla or at Erb's point) is incapable of producing contraction of the extensors of the forearm, whereas it is effective when the electrode is applied below the point of compression. In this case the radial nerve, although its excitability is preserved, has lost its power of conducting voluntary impulses and of transmitting an electrical stimulus. Conversely, when a nerve which has lost its motor functions and electric excitability begins to recover these properties, the earliest phenomenon is usually recovery of the power of voluntary movement, after which the nerve above the lesion becomes excitable, and later still, excitability below the lesion is recovered. Here again there is therefore dissociation of excitability and conductivity.

These two different properties of the nerve, excitability and conductivity, are usually attributed to two different elements in the neurone. The function of conductivity, both voluntary and electrical, is concerned with the axis cylinder, while the function of excitability is connected with the myelin sheath, and it is by the myelin that electric stimuli are transmitted.

Now, in nerve lesions it appears that changes in the myelin precede those in the axis-cylinder.

At the same time the axis-cylinder should apparently recover its anatomical integrity earlier than the myelin, since in the process of regeneration a nerve recovers its power of conducting voluntary impulses earlier than its excitability.

IV. Stimulation with the Faradic Current

Single Faradic Contractions from Induction Shocks (fig. 12).—When a motor nerve or muscle is stimulated by means of an induced current progressively increasing in intensity, produced at break of the interrupter, it is found that with a low intensity only the wave induced at break produces an effect. To obtain a contraction with the wave induced at make, the intensity must be increased considerably, and with such a current the break contraction is decidedly more forcible than the make.

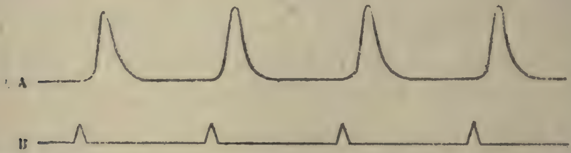

Fig. 12.—Single faradic contractions (A). At B the induction shocks (waves at break).

The greater intensity and shorter duration of the break wave are the two physical factors which explain this difference between the make and break waves.

The total duration of the muscular contraction being about one-tenth of a second, a succession of single contractions will be obtained by adjusting the interrupter for a lower frequency than 10 stimuli per second. In practice, however, to obtain clearly distinct contractions, there must be an interval of rest between each at least equal to the time of the contraction. The contractions are only clearly isolated with a frequency of 1 to 2 stimuli per second.

Physiological Tetanus.—When, on the other hand, a stimulus reaches the nerve or muscle during the period of relaxation after a previous contraction, a second contraction prolongs the first and increases its amplitude. A succession of stimuli applied under these conditions produces *incomplete physiological tetanus* (fig. 13). This phenomenon is given by frequencies of from 3 to 15 interruptions per second.

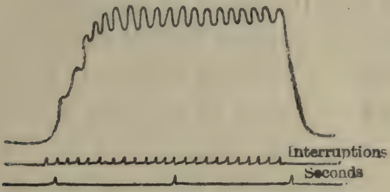

FIG. 13.—Incomplete faradic tetanus.

By adjusting for higher frequencies, a point is reached at which the stimuli recur with sufficient rapidity to coincide with the end of the contraction period caused by the preceding stimulus. Hence the muscle has no longer any time to relax; in spite of the succession of stimuli, it displays the picture of a single contraction. This is spoken of as fusion of contractions, or *complete physiological tetanus* (fig. 14).

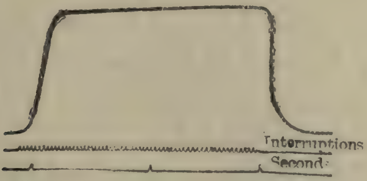

FIG. 14.—Complete faradic tetanus.

A further increase of frequency beyond that producing complete tetanus only increases the amplitude of the contraction.

The minimum frequency to produce tetanus varies with the subject and with the muscle. As a rule, it is given by a frequency of 18 to 25.

Current of Quantity and Current of Tension.—It is usual to say that the secondary current produced in the coil with fine wire (current of tension) acts on the sensory apparatus, whereas current from a coil with coarse wire (current of quantity) acts on the motor apparatus. These expressions suggest a specificity which does not exist. It is certain that coils with fine wire, particularly when the current is applied by means of dry electrodes, have a marked effect on sensory nerve-endings, and the more the tension is raised the more painful does the current become. With moist electrodes the sensory effect is less, but it is always more marked than with the current of quantity. With the coil of coarse wire and with moist electrodes, pronounced motor effect can be developed by approximating the coils, without causing painful sensation. If, however, a contractile response is not obtained at the maximum approximation, the intensity must be insufficient. A contraction can then be obtained with the finely wound coil, which owing to the greater tension of the current will give a greater intensity. In certain cases this contraction may, instead, be obtained by using a coil giving a current of lower tension (medium winding), which consequently will produce only a moderate degree of painful sensation.

V. STIMULATION WITH THE GALVANIC CURRENT

The Law of Contraction.—The stimulation of a nerve or muscle by a continuous current in the permanent state is ineffective with low intensities of current. With adequate intensities there results, not a muscular contraction, but an increase of the normal tone (Remak's galvanotonic contraction). To cause contractions with the galvanic current it is necessary to produce sharp variations of intensity (Du Bois-Reymond).

In experimental physiology, when two electrodes are applied to an exposed motor nerve, and a current of moderate intensity is sharply made and broken, a twitch is observed in the muscles innervated by the nerve excited, at the instant of sudden make and sudden break.

If the intensity of a constant current already traversing the nerve is suddenly diminished or increased, the same phenomenon is observed.

The contraction at make and that at break, however, only occur with currents of moderate intensity. With weak currents, sufficient to produce only one contraction, the twitch only occurs at the make; again, with strong currents only one twitch is observed, which takes place at the make if the current is descending, and at the break if it is ascending.

These peculiarities constitute what is known as

Pflüger's law of contraction. But although this law is regarded as one of the fundamental discoveries of electrophysiology, it is now only of secondary interest from the clinical point of view, by reason, in particular, of the fact that it is almost impossible, unless very strong currents are used (Brenner), to reproduce the conditions of Pflüger's experiment through the skin in man.

The Unipolar Method of Stimulation. — Pflüger's law regards the direction of the current, whether ascending or descending, as the essential factor in determining the order of the muscular responses.

It was subsequently questioned, however, whether these phenomena really depended on the direction of the current, or whether they did not follow simply from the specific action of each pole. To decide this question, a means must be found of studying their respective actions separately.

The unipolar method of stimulation, introduced in physiology by Chauveau, and shortly afterwards by Brenner as a clinical method, apparently proved the specificity of each of the poles.

In this method, the nerve is investigated by stimulation with *a single electrode*; the other, known as the *indifferent or passive electrode*, being fixed at a point some distance from the nerve concerned.

The Order of Appearance of Contractions: The Normal Formula.—In man, the effects of unipolar stimulation may be studied by applying a large indifferent electrode to some point, preferably the patient's back, and applying a tampon electrode, 2 sq. cm. in area, over an easily accessible nerve. Under these conditions, if the intensity of the current be gradually increased, the following phenomena are observed.

At first no contraction is produced until the threshold intensity is reached; this varies with different nerves from 0·1 to 5 milliamperes.[1] Then, with a weak current, and the active electrode connected to the negative pole, the varying state of make produces a *short, sharp, and rapid* contraction, while the break has no effect. The same stimulus with the same strength of current, but with the positive pole in connection with the nerve, produces no contraction, either at make or at break.

With a stronger current, and the active electrode connected to the negative pole, the make gives a

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[1] The degree of intensity required to excite different nerves varies with the nerve concerned, the regulating mechanism, the surface area of the active electrode, etc. Thus with electrodes 3 sq. cm. or more in area, a rather stronger current is required to obtain the threshold than with smaller instruments. Adjusting the intensity with a rheostat, or a resistance introduced into the stimulating circuit, gives higher values for the threshold than are obtained with a potential reducer or a cell-collector. Lastly, there may be very wide individual variations. Usually, with a 1 to 2 sq. cm. electrode, and a collector, the threshold is as follows :

Musculo-cutaneous . . .	0·1 ma. to 0·3 ma.
Ulnar (in the sulcus) . . .	0·5 ,, ,, 2·5 ,,
,, (in the arm) . . .	0·2 ,, ,, 1·0 ,,
Median	0·3 ,, ,, 1·5 ,,
Radial	1·0 ,, ,, 3·0 ,,
Facial	0·5 ,, ,, 2·0 ,,
Crural	0·5 ,, ,, 2·0 ,,
External popliteal . . .	0·2 ,, ,, 2·0 ,,

more forcible contraction than before: no twitch occurs at the break. If the active electrode is made positive, a current of the same intensity produces: (1) a fairly strong contraction at make, (2) a weaker contraction at break.

With a still stronger current, and the active electrode negative, we obtain, (1) a persistent tetanic[1] contraction at make, (2) a slight twitch at the break. The latter is only perceptible with a very strong current, which is often painful.

If the active pole is positive, contractions in the same ratio as in the preceding case occur at make and break, both, however, with considerable force.

In observing the intensities at which these different contractions appear, they are seen to succeed one another in the following order: first KCC, then ACC, then AOC, and lastly KOC; this is usually represented by the conventional formula:

$$\mathrm{KCC} > \mathrm{ACC} > \mathrm{AOC} > \mathrm{KOC};$$

it is sometimes written $\mathrm{KCC}te > \mathrm{ACC} > \mathrm{AOC} > \mathrm{KOC}$, to indicate the tonic nature of the kathodal closing contraction when strong currents are employed.

In reality, as Cardot and Laugier point out, this formula indicates, not the order of appearance of these stimuli, but the magnitude of the contractions with a current of sufficient intensity to produce all four of them. The exact formula of their order of appearance would be:

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[1] We use the word tetanic here out of respect for the old terminology. But as Bourguignon very rightly said, the word tetanus has a definite meaning in physiology, suggesting the idea of a fusion of simple contractions. Now the galvanic current produces a simple, sustained, and tonic contraction; that is, a galvanotonic contraction.

$$\text{Threshold KCC} < \text{threshold ACC} < \text{threshold AOC} < \text{threshold KOC.}$$

This formula is the general rule. With certain nerves, however, the positive break contraction sometimes appears with an intensity equal to, or less than, that necessary for obtaining the positive make contraction. This peculiarity, already described by Remak and Erb, is not infrequently observed in the radial, the external popliteal, and sometimes the median and ulnar nerves. The formula then becomes:

$$\text{KCC} > \text{AOC} \geqq \text{ACC} > \text{KOC.}$$

When, instead of applying the active electrode to the nerve it is applied *over the muscle*, we observe that the order of appearance of the contractions is the same. The formula is identical, with the following modifications:

1. A current of liminal intensity for the muscle causes a relatively powerful contraction if applied to the nerve; the latter is therefore more excitable than the muscle.

2. Break contractions require a greater intensity on the muscle than on the nerve: such an intensity as may become painful. The KO contraction should

not, at times, be produced, since the current necessary to produce it would be unbearable.[1]

3. The galvanotonic contraction KCC*te* is much more difficult to obtain than on the nerve.

This order of appearance of contractions is common to man and animals. In animals, however, the results are no longer the same when, instead of determining the smallest intensity of current necessary to provoke contraction, currents of increased intensity are applied. Instead of the predominance of KCC over ACC, it is often observed that KCC and ACC are equal, or even ACC may be greater than KCC.

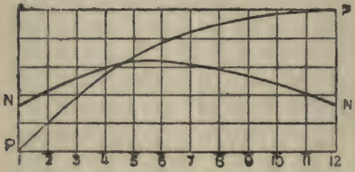

FIG. 15.—Variation in amplitude of contraction produced by each pole as determined by the intensity of the current (Chauveau).

Chauveau has shown, in fact, on the frog, that with regularly increasing unipolar excitations, the negative response increases in amplitude and duration, at first quickly, then more slowly, whereas the positive increases in a regular and constant manner up to the maximal contraction. It follows that for a certain intensity, the contractions are equal; the ACC becomes equal in force and amplitude to the KCC (Chauveau's neutral point) (fig. 15). With still greater intensities, the ACC predominates entirely, contractions with the negative pole remaining stationary or even beginning to weaken. Thus an actual inversion occurs. According to Boudet of Paris, if the strength of the current be increased still further, contractions with the positive pole diminish in their turn, then again equalise those from the negative pole, and finally diminish more rapidly than the latter. There would therefore be a second neutral point.

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[1] The AOC is obtained with relative ease on such large muscles as the biceps, supinator longus, flexor digitorum, vastus internus, sterno-mastoid, and trapezius. But with small muscles such as those of the face, more than 5 ma. are sometimes required in the normal state, and produce very painful sensations. The KO contraction is naturally still more difficult to obtain.

With break currents the opposite phenomenon occurs.

In the normal state in man, it is difficult to demonstrate this phenomenon, as a current of sufficient strength cannot be used. But in a pathological state, this is not so, as will be seen later (Bourguignon).

The Supposed Specific Action of the Poles.—Results of the unipolar method of investigation possess the value belonging to observed facts. Each of the poles, at make and at break, has a different exciting power.

It is for this reason that in electro-diagnosis a very large indifferent electrode is used on the one hand, and on the other, an active electrode transmitting the current at a very high density, a procedure by which the effect of one of the poles is absolutely excluded, through an increase in the exciting power of the one of which the action is being studied.

This supposed polar specificity is, however, only apparent. It is explained in the law of electrophysiology stated simultaneously by Pflüger, von Bezold, and Brenner (1859): *stimulation originates at the kathode at make, at the anode at break.* Brenner, then de Watteville, Erb, and finally Bourguignon, proceeded to demonstrate that this law, based on experiments with an animal, by the bipolar method, was true for man, and with the unipolar method.

At the make, when the testing electrode is negative, the stimulus originates from it; when it is positive, the stimulus passes from the indifferent electrode, and impinges on the nerve or muscle over a much wider area (virtual electrode), and consequently at a much lower density. This reduced density can only produce a contraction of a lesser intensity. At the break, the negative pole being on the nerve or muscle, the stimulus passes from the indifferent electrode, and reaches the nerve or muscle at a lower density than would be the case were the positive electrode applied to the organ concerned. Hence it follows that the AOC should be greater than the KO contraction.

In reality, when we stimulate a nerve or muscle alternately with negative and positive, we are not bringing a selective action of each pole into play, after excluding effects from the pole of opposite name. We simply effect stimulation of the organ at a density which varies according to the point of origin of the stimulus.

VI. Exploration

General Technique.—To carry out an electro-diagnostic examination under good conditions, the region to be explored should be placed in a good light, and in such a position that the muscles to be stimulated are in a state of complete relaxation. For this reason, it is often more convenient to examine the patient lying on a bed or a couch.[1]

It is always well, and all classical works are agreed on this point, to commence with faradic exploration, first the nerve and then the muscle. Beginners will do well to follow this rule, but experienced electrologists prefer to begin with the galvanic exploration of muscles. This, owing to the form of the contraction and the strength of the current required, enables conclusions to be formed rapidly. It shows immediately, at the first touch of the key, whether the reaction of degeneration is present or not, and subsequent faradic exploration completes this original observation by indicating whether the reaction of degeneration is complete or partial.

Finally, the passage of the continuous current sometimes reveals latent faradic excitability in the muscle (*v.* p. 81). By beginning with galvanic exploration, it will be possible to decide more definitely whether faradic excitability is absent or preserved, which is of the greatest importance from the point of view of the degree of the reaction of degeneration.

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[1] It is often important, for reasons which will be explained on p. 80, to preface an examination by immersing the limb concerned for some time in warm water.

The examination of muscles with the galvanic current should concern itself with the nature of the contraction, the minimum strength required to obtain the threshold, and the order of appearance of the contractions. As a rule the break contractions are not studied; the characteristics of the make contractions alone usually suffice to indicate the degree of hypo- or hyper-excitability, the form of the contraction, and the existence or absence of the inversion reaction.

When the galvanic examination of the muscles has been completed, the faradic examination is undertaken; finally, the nerves are examined.

Either a frequently interrupted faradic current, or single shocks, will be used, according to circumstances (*v.* p. 53). Whenever unilateral lesions are concerned, the nerves and muscles of the unaffected side should be examined from time to time, as their normal reactions are of the utmost value for comparison.

Unipolar Method.—The electrodes by which the current is to be transmitted to the patient are located according to the principle of the unipolar method: one, the indifferent electrode, at some point on the dorsal or lumbar region, or in front of the sternum (in such a way that the sagittal plane of the body cuts it into two equal parts); the other, the testing electrode, upon the nerve or muscle to be examined. The latter will be chosen with suitable diameter:

small (8 to 10 mm. in diameter) for the examination of the nerves and muscles of the face, the small muscles of the hand; medium (2 to 2·5 cm. in diameter) for the examination of muscles of moderate size; and larger for thick muscles, such as those of the calf, or the quadriceps femoris.

Bipolar Method.—It often happens that when the faradic excitability of a muscle is much diminished, the strong faradic stimulus employed to produce contraction is conducted to the healthy muscles of the region. In this case the limb under examination is violently shaken, and the contraction of neighbouring muscles obscures that of the muscle under

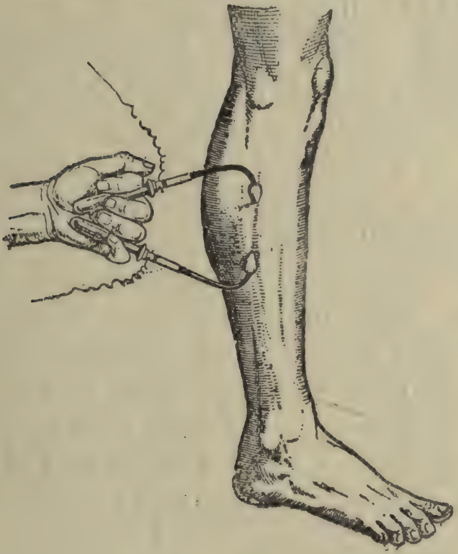

FIG. 16.—Bipolar method.

observation, the excitability of which cannot, therefore, be estimated.

The indication then is to localise the action of the current to this muscle. For this purpose the wires from the coil are connected with two small electrodes of suitable dimensions, preferably small olive-shaped electrodes, which are applied approximately over the region of the ellipse formed by the fleshy belly of the muscle. This is the bipolar method, which Duchenne (of Boulogne) found so satisfactory (fig. 16).

With the galvanic current, the examination should always, as a matter of principle, be carried out by the unipolar method. When, however, the excitability of the region under examination is much reduced, stimulation, owing to the great strength of current required, again provokes violent contractions in neighbouring regions which are unaffected by the lesion. It is therefore sometimes of advantage to bring the indifferent electrode close to the region under examination, to fix it at the root of the limb, or again at a short distance above the muscular area concerned.

To avoid the masking of the reactions of the muscles affected with the reaction of degeneration by those of healthy muscles, produced by a strong current, Larat advises gentle rubbing of the muscle under examination with the testing electrode, and gradually increasing the strength of the current until a response is obtained. At the moment when the electrode comes in contact with the skin, the healthy muscles undergo a sharp, rapid, and only momentary contraction, followed by the slow contraction of the degenerated muscle.

More scientific, but yet outside the domain of practice, is the arrangement, devised by Lapicque, of a condenser placed *in parallel* with the stimulating circuit, the latter being rendered more resistant by an additional resistance, intended to retard the making of the current. The galvanic wave is thus rendered too slow to excite healthy *striped* muscle, and only provokes the slow contraction of the degenerated muscles.

Motor Points.—Trans-cutaneous stimulation is only practicable when the nerve or muscle is superficially situated, *i.e.* only a short distance beneath the skin. Deep nerves and muscles cannot be investigated.

Nerves are not usually accessible except in their exposed parts, at points where they are not surrounded by muscle. (Example: The median nerve between the flexor tendons above the wrist, the ulnar in the epitrochlear-olecranon sulcus.)

Sometimes, however, it is difficult to reach them, as in a case where the nerve runs alongside the body of a muscle. Here stimulation causes a simultaneous contraction of this neighbouring muscle, which, in expanding, often intervenes between the electrode and the nerve. (Example: The radial nerve in the lower part of the musculo-spiral groove.)

All the fleshy part of a *muscle* is excitable, but this excitability is always more pronounced over a limited area, which generally corresponds both to the most superficial part of the muscle, and to the point of entry of the nerve which supplies it. These areas where nerves are accessible, or where muscles can most readily be excited, are called *points of election*, or *motor points*.

Muscles usually possess *one* motor point, but a few multi-fascicular muscles have *two*, or even three, like the deltoid.

It is with these points that the exploring electrode is concerned.

All works on electrotherapy contain plates showing the topography of the motor points; that is, their position on the surface of the skin. The electrologist, however, should know the situation of these points sufficiently well by heart to dispense with frequent reference to diagrams, which complicates an examination considerably.

To acquire a rapid familiarity with their situation it is much more profitable, in our opinion, to use good anatomical plates than to study the diagrams referred to above.

A mental dissection of the region to be examined suggests the relations of the parts, and assists the memory of these points of election, giving the hand which is manipulating the electrode that assurance and precision required for their "discovery."

Lastly, it must be remembered that the classical points are subject to individual variations in position, so much so that the motor point is often discovered only after a succession of tentative stimuli over a given area.

The classical plates all show an inordinately large number of motor points. In addition to those which it is absolutely necessary to know, there is a whole series of points which are never examined, or only in such exceptional conditions[1] that it seems to us altogether useless to reproduce them here, particularly as the situation of the principal ones will be all the more evident in consequence. Finally, we have definitely broken with old tradition in diagrams of motor points by showing, as in works on operative medicine, such anatomical relations as will enable the operator to locate these points with ease and rapidity.

THE PRINCIPAL MOTOR POINTS[2]

THE MOTOR POINTS OF THE FACE

Examination of the muscles of the face should be carried out with a small electrode, less than 1 *cm. in diameter. The faradic current is only employed in the form of single shocks, as far as possible of low tension, in order to reduce stimulation of sensory nerve-endings to a minimum.*

1. **Facial Nerve.**—The facial nerve is stimulated either below the ear, between the mastoid and the ascending ramus of the mandible, the electrode being applied as if with the object of reaching the stylo-mastoid foramen, or in front of the ear, at the base of the tragus. It may also be reached by inserting a small conical electrode into the external auditory meatus, and moving this electrode downwards and forwards (Bergonié). Stimulation of the facial nerve causes contraction of all the muscles of the face.

The three branches of the facial nerve may be stimulated separately.

(1) Upper branch : on the temple, slightly outside and above the outer angle of the eye.

(2) Middle branch : over the most prominent point of the zygomatic arch.

(3) Lower branch : immediately above the angle of the lower jaw and slightly in front of it.

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[1] As, for instance, the procerus (*pyramidalis nasi*) and nasalis in the face, the supinator brevis and anconæus in the arm, the tensor fasciæ latæ and pectineus in the thigh, etc.

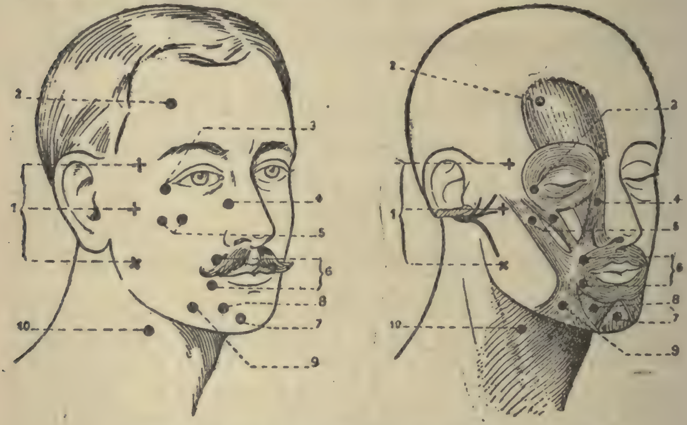
[2] The numbers correspond to those in the illustrations.

These three points are situated in an almost straight and vertical line passing through the most prominent point of the zygomatic process.

Care should be taken, in examining branches of the facial nerve, not to be led into error by contractions of the masseter and temporalis muscles (trigeminal), to which the stimulus is conducted when a current of sufficient strength is used.

2. Frontalis muscle.—*Motor point :* over the prominence of the frontal bone,[1] above the external region of the orbit. Elevates the eyebrow and furrows the forehead.

3. Orbicularis oculi.—*Motor point :* below the external angle of the eye. Closes the eyelids.



Figs. 17 and 18.—Motor points of the face.

4. Levator labii superioris alæque nasi.—*Motor point :* over the lower border of the nasal bone. The action is indicated by its name.

5. Zygomaticus major and minor.—*Motor point :* below the lower border of the malar bone, behind the insertion of the zygomatic process.

Draw the labial commissure upwards and outwards.

6. Orbicularis oris.—*Motor points :* two distinct points, for the upper and lower parts of the muscle, above and below the labial commissure. (During the examination of any muscles situated round the mouth, the latter should be slightly open.)

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[1] Stimulation of the frontalis often causes phosphenes or giddiness.

Sphincter closing the lips.

7. LEVATOR MENTI.—*Motor point:* on the lower border of the mandible, just outside the middle line.

Draws the lower lip downwards and forwards, furrows and expands the chin.

8. DEPRESSOR LABII INFERIORIS.—*Motor point:* lateral to the foregoing, and slightly higher.

Draws the lower lip downwards and outwards.

9. DEPRESSOR ANGULI ORIS.—*Motor point:* immediately above the lower border of the mandible, at the junction of the posterior two thirds and anterior third of the horizontal ramus.

Draws the labial commissure downwards and outwards; antagonises the zygomaticus major.

10. PLATYSMA.—*Motor point:* below and slightly anterior to the angle of the mandible. (It is important to differentiate between contractions of this muscle and those of the sterno-mastoid. For this reason the electrode is only applied very lightly to the skin, and not pressed deeply into it.)

Stretches the skin of the neck, and slightly depresses the labial commissure.

BUCCINATOR.—*Motor point:* within the mouth, in front of the anterior border of the masseter.

This muscle draws the cheeks inwards and the labial commissure outwards.

MOTOR POINTS OF THE SPINAL ACCESSORY NERVE AND CERVICAL PLEXUS

11. **Spinal Accessory Nerve** (EXTERNAL RAMUS).—*Motor point:* at the upper angle of the supra-clavicular triangle.

Causes contraction of the sterno-cleido-mastoid and trapezius (clavicular portion).

12. STERNO-CLEIDO-MASTOID.—*Motor point:* at the junction of the upper and middle third of the muscle.

Flexes the head, bends it to the side excited, and turns the face towards the opposite side.

13. TRAPEZIUS, CLAVICULAR PART (SPINAL ACCESSSORY NERVE.—*Motor point:* on the anterior border of the muscle, the edge of which is easily felt, and towards the middle of its clavicular portion.

Stimulation of the clavicular portion bends the head to the side stimulated, and slightly backwards, while the face is turned towards the opposite side.

14. MIDDLE PORTION (CERVICAL PLEXUS).—*Motor point:* in the vertebral groove towards the inner angle of the scapula. Raises the scapula.

15. LOWER PORTION (CERVICAL PLEXUS).—*Motor point:* below the foregoing, at the level of the hollow of the axilla.

Draws the scapula towards the spine and carries the shoulder backwards.

RHOMBOIDS (CERVICAL AND BRACHIAL PLEXUS).—*v.* BRACHIAL PLEXUS, p. 40.

MOTOR POINTS OF THE UPPER LIMB

(1) Upper Radicular Group, and collateral branches of the Brachial Plexus

16. **Erb's Point.**—Two or three cm. above the clavicle, slightly outside the lateral border of the sterno-mastoid, opposite the transverse process of the sixth cervical vertebra (Chassaignac's tubercle).

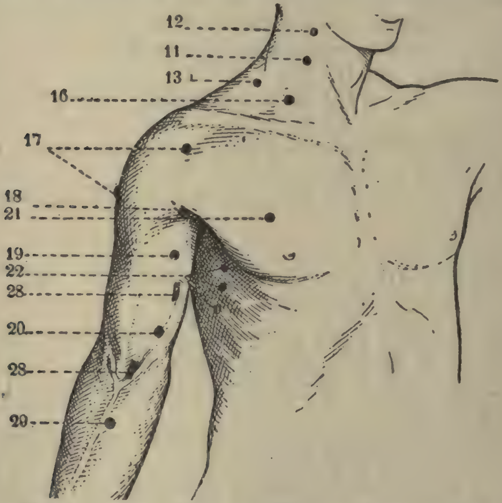


FIG. 19.—Motor points of the shoulder and arm (anterior aspect).

Stimulation of Erb's point causes simultaneous contraction of the four muscles of the Duchenne-Erb group; and of the pectoralis major (Secrétan, Mme. Klumpke). In total radicular paralyses of the superior type, stimulation at Erb's point gives rise to no contraction whatever. The build of the shoulder sometimes modifies the situation of this point appreciably.

17. DELTOID.—*Motor points:* on the anterior surface of the muscle approximately in the upper third; a second on the posterior

surface at the same level, and a third, stimulation of which causes contraction of the external fasciculi.

The anterior fasciculi raise the arm and carry it forward, the posterior carry it backwards.

18. **Musculo-cutaneous Nerve.**—*Motor point:* very difficult to find and to isolate; the electrode must be pressed very firmly between the two heads of the biceps, or better between the coraco-brachialis and the biceps.

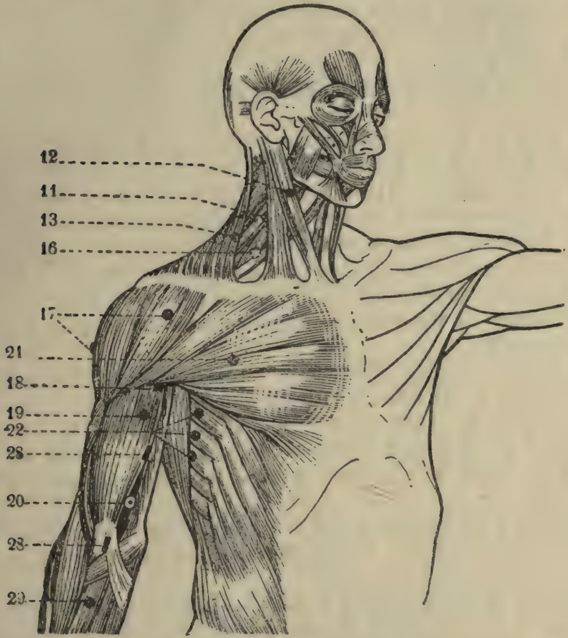


FIG. 20.—Motor points of the shoulder and arm (anterior aspect).

Causes contraction of the biceps and brachialis.

19. BICEPS.—*Motor point:* slightly below the antero-inferior border of the deltoid.

Flexes and supinates the forearm.

20. BRACHIALIS.—*Motor point:* to excite this muscle, a small electrode must be insinuated beneath the internal border of the biceps slightly below the middle of the arm, care being taken to avoid the median nerve. As a rule it is very difficult to isolate, since the

biceps or the median and ulnar nerves respond simultaneously to the stimulus and obscure the movement produced. It is sometimes easier to stimulate outside the biceps, between this muscle, the triceps and the upper border of the supinator longus. The flexors of the forearm must be kept in a state of complete relaxation.

Flexes the forearm on the arm.

SUPINATOR LONGUS (see motor points of the Radial nerve).

21. PECTORALIS MAJOR.—*Motor point :* above and lateral to the nipple on an imaginary line prolonging the anterior border of the hollow of the axilla.

Draws the arm downwards from a raised position, and rotates it internally.

22. SERRATUS MAGNUS.—*Motor points :* each digitation has a motor point. These are situated in the anterior part of the axilla along an oblique line with a posterior inclination from above downwards. Contraction of the entire muscle can be obtained by stimulating the nerve to the serratus magnus, which can be effected on the thoracic wall near the apex of the axilla, the arm being suitably raised in order to displace the pectoralis major.

Contraction of the serratus magnus draws the scapula forwards and outwards.

23. TERES MAJOR.—*Motor point :* this muscle connects the internal border of the scapula with the inner lip of the bicipital groove of the humerus. It forms part of the posterior wall of the axilla. Its motor point should therefore be looked for on the posterior wall of the axilla, near the apex of the latter. This muscle is difficult to isolate.

It adducts the humerus and raises the point of the shoulder.

24. SUPRASPINATUS.—*Motor point :* in the external angle of the supraspinous fossa.

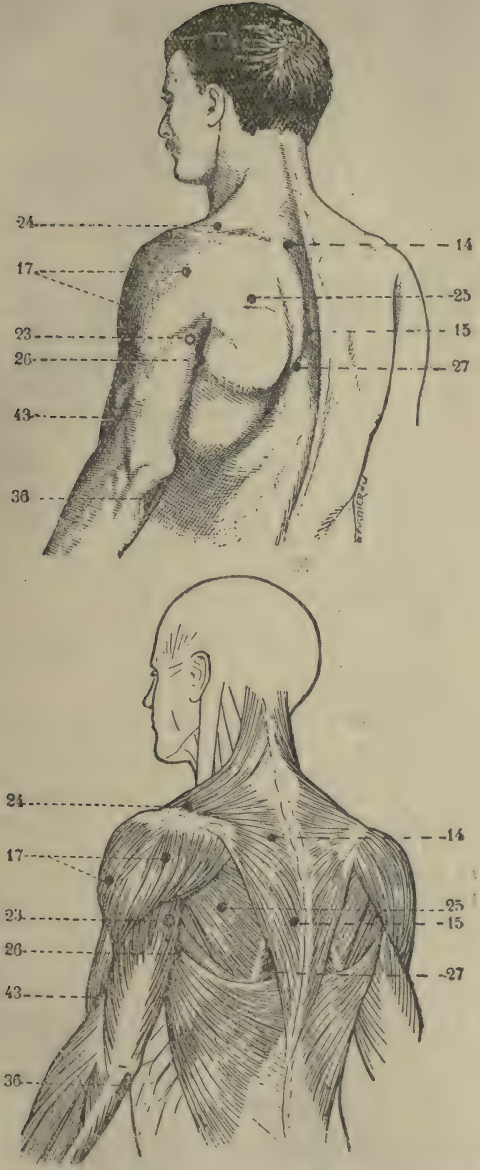
This muscle lies beneath the trapezius and can only be stimulated with great difficulty. It is only accessible when the trapezius is atrophied.

25. INFRASPINATUS AND TERES MINOR.—*Motor point :* the posterior border of the deltoid and the external border of the trapezius on separation bound a triangular aperture, the base of which is formed by the latissimus dorsi : it is through this aperture that the infraspinatus and teres minor are stimulated.

Rotate the arm externally.

26. LATISSIMUS DORSI.—*Motor point :* on the posterior wall of the axilla, between the scapula and the thoracic wall; it adducts, extends, and internally rotates the humerus, draws the scapula inwards towards the middle line, and the shoulder downwards.

27. RHOMBOIDS.—*Motor point :* these muscles can be stimulated in



FIGS. 21 and 22.—Motor points of the shoulder (posterior aspect).

the triangle bounded by the lower border of the trapezius, the vertebral border of the scapula, and the upper border of the latissimus dorsi.

Raises the internal angle of the scapula and draws it towards the spine, rotating it on the external angle of the bone.

(2) Median and Ulnar Nerves

28. **Median Nerve.**—*Motor points :* in the arm, along the internal border of the biceps, but as a rule simultaneous excitation of the ulnar and musculo-cutaneous cannot be avoided while stimulating at this point ; at the elbow, in the flexion fold medial to the biceps tendon ; at the wrist, at the level of the flexion folds between the tendons of the palmaris longus laterally and the palmaris brevis medially.

Stimulation at the elbow causes forcible pronation of the forearm, with flexion and closing of the hand (flexion of the fingers, opposition of the thumb) ; at the wrist, stimulation causes abduction of the hand and opposition of the thumb.

29. PALMARIS LONGUS.—*Motor point :* in the upper third of the muscle, on the ulnar border of its fleshy portion.

Flexes the hand.

30. PRONATOR TERES.—*Motor point :* on its external border in the upper third.

Causes pronation of the hand.

31. FLEXOR DIGITORUM SUBLIMIS.—*Motor points :* flexion of the last two fingers can be obtained by stimulation at a point towards the middle of the forearm, medial to the tendon of the palmaris longus. The point for the flexor of the index is in the lower third of the forearm, slightly within the middle line ; and just medial to this is the point for the middle finger.

Flexes the second phalanges of the four last fingers.

32. FLEXOR DIGITORUM PROFUNDUS.—*Motor point :* difficult to find ; separate stimulation almost impossible ; in the middle of the forearm, between the palmaris longus and flexor carpi ulnaris, towards the inner surface of the ulna.

Flexes the terminal phalanx of the last four fingers.

33. ABDUCTOR POLLICIS BREVIS.—In fig. 25 the electrode is shown on the motor point of this muscle. It will be observed that this point is proximal and lateral, on the outer border of the thenar eminence, close to the dorsal surface of the hand.

Carries the thumb forwards and inwards.

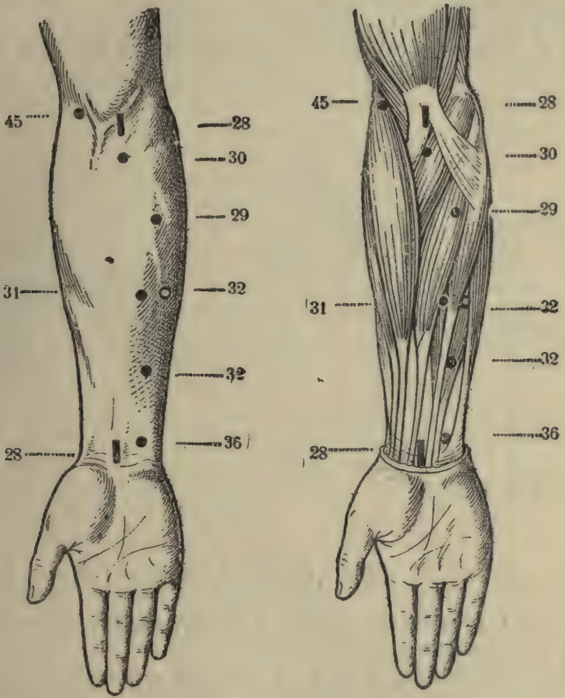
34. FLEXOR POLLICIS BREVIS.—*Motor point :* towards the internal border of the thenar eminence. It should not be forgotten that

this muscle consists of two parts, one supplied by the median, and the other by the ulnar nerve.

Draws the thumb inwards and forwards, and slightly rotates the first metacarpal in its long axis.

35. Opponens pollicis.—*Motor point:* slightly proximal and medial to the foregoing.

The three muscles of the thenar eminence should be stimulated with a very small electrode, such, for instance, as that used for the muscles of the face.



Figs. 23 and 24.—Motor points of the forearm (anterior aspect).

36. **Ulnar Nerve.**—In the arm: to avoid simultaneous stimulation of the median, the electrode should be applied about two fingers' breadths above the epicondyle.

At the elbow: the electrode is pressed into the epitrochlear-olecranal sulcus.

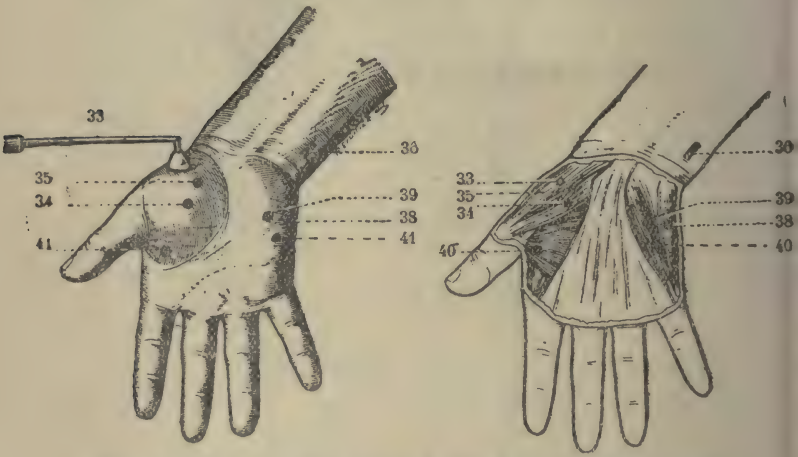
At the wrist: above the skin-folds and immediately lateral to the tendon of the flexor carpi ulnaris.

Stimulation at the elbow produces ulnar contraction of the fingers and flexion and adduction of the hand. Stimulation at the wrist draws the hand medially, and causes flexion of the three last fingers and adduction of the thumb.

37. Flexor carpi ulnaris.—*Motor point:* two fingers' breadths below the internal epicondyle.

Flexes and adducts the hand.

38. Abductor minimi digiti.—*Motor point:* on the ulnar border of the hand, towards the middle of the hypothenar eminence (internal to the pisiform).



Figs. 25 and 26.—Motor points of the hand (palmar aspect).

Abducts the last finger from the long axis of the hand.

39. Flexor brevis minimi digiti.—*Motor point:* in the centre of the hypothenar eminence (internal to the foregoing, and immediately distal to the hook of the unciform bone).

Flexes the last phalanx of the little finger and extends the two other phalanges.

40. Opponens minimi digiti.—*Motor point:* slightly below and external to the foregoing; difficult to isolate.

41. Adductor pollicis.—*Motor point:* on the internal side of the first metacarpal space (palmar aspect).

Abducts the first metacarpal and flexes the first phalanx.

42. DORSAL INTEROSSEI.—*Motor points :* at the distal ends of the inter-metacarpal spaces on the dorsum of the hand.

Flex the first phalanx and extend the two others. A more characteristic movement, however, is approximation of the two fingers adjacent to the interosseous space where the stimulus is applied.

(3) Radial Nerve

43. **Radial Nerve.**—The point for stimulation is often difficult to find. In order not to miss it a line should be imagined drawn from the middle of the posterior border of the deltoid V to the epicondyle. This line corresponds to the inter-muscular septum. In an adult a distance of approximately 10 cm. above the epicondyle is measured. This point is between the outer head of the triceps and the brachialis, above the first insertions of the supinator longus, at the point where the radial nerve perforates the septum.

It is well to use a fairly small electrode, which can be pressed deeply in between the brachialis and the most anterior fasciculi of the outer head of triceps. The radial may also be stimulated in the axilla, but it is difficult to avoid exciting other nerves in this region. In common radial paralyses, stimulation at this point may give rise to a false interpretation of the electrical reactions. Conductivity being interrupted below the point of pressure, faradic stimulation in the axilla only causes contractions in the triceps (the nerve to which rises above this point), and not in the extensors. When the electrode is applied on the humeral point, the latter muscles will be observed to respond to stimulation.

44. TRICEPS.—*Motor points :* for the long head, over this part of the muscle, in its upper third.

For the outer head : in the lower third of the external aspect of the arm.

For the inner head : in the centre of the internal aspect of the arm.

The triceps is the extensor of the forearm.

45. SUPINATOR LONGUS.—*Motor point :* on the anterior border of the muscle, in the skin-fold at the elbow.

Flexes the forearm in semi-pronation.

46. EXTENSOR CARPI ULNARIS.—*Motor point :* on the internal border of the ulna in its upper third, slightly below the olecranon.

This muscle extends the wrist and draws the hand inwards.

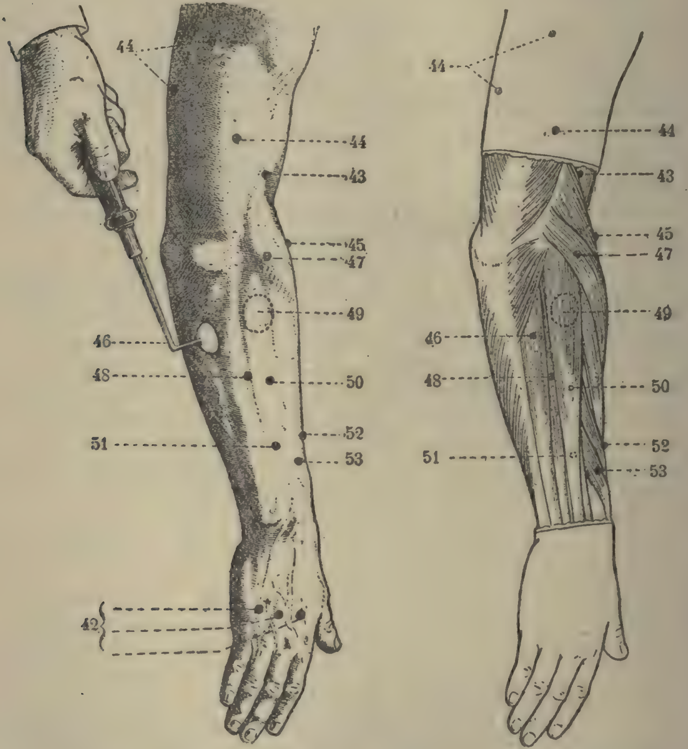
47. EXTENSOR CARPI RADIALIS LONGIOR.—*Motor point :* on the upper and outer part of the dorsal aspect of the forearm.

Extends the wrist and draws the hand outwards.

48. EXTENSOR MINIMI DIGITI.—*Motor point :* slightly distal and lateral to the point for the extensor carpi ulnaris.

49. **Extensor digitorum communis.**—*Motor point :* in the upper third of the dorsal aspect of the forearm.

An electrode of considerable size is required for stimulating all parts of the muscles simultaneously. Each can always be stimulated separately if it is remembered that the points for the middle and index fingers are situated at the same level as the extensor of the little finger, and lateral to it, and those for the ring finger a finger's breadth above and slightly external to the latter.



Figs. 27 and 28.—Motor points of the forearm and hand (dorsal aspect).

This muscle extends the first phalanx on the metacarpal, and the hand on the forearm.

50. Extensor indicis proprius.—*Motor point :* on the external border of the ulna, in its lower third.

51. Extensor pollicis longus.—*Motor point :* in the middle of the dorsal aspect of the forearm, four fingers' breadths above the line of articulation of the wrist. The electrode is pressed firmly on this point.

52. Abductor pollicis longus.—*Motor point :* on the external border of the radius, where the muscle forms a prominence.

53. Extensor pollicis brevis.—*Motor point :* slightly to the inner side of and below that for the abductor longus.

Extends the first phalanx of the thumb on the metacarpal and abducts the thumb.

MOTOR POINTS OF THE LOWER LIMB

(1) Anterior Crural Nerve

54. **Anterior Crural Nerve.**—The point for this nerve is below and slightly internal to the middle point of the inguinal ligament.

Stimulation causes forcible extension of the leg on the thigh.

55. Sartorius.—*Motor point :* in the upper third of the muscle or towards the middle.

Stimulation does not usually produce the physiological movement of flexion of the thigh on the pelvis, of the leg on the thigh, and lateral rotation of the thigh. It is sufficient to detect contraction of the muscle beneath the skin.

56. Rectus femoris.—*Motor point :* slightly above the middle of the anterior surface of the thigh.

Extends the knee, and draws the patella upwards.

57. Vastus internus.—*Motor point :* to the inner side of the inner border of the rectus femoris, four fingers' breadths above the patella.

Draws the patella upwards and inwards.

58. Vastus externus.—*Motor point :* lateral to the external border of the rectus femoris, and slightly higher than that of the preceding muscle.

Draws the patella upwards and outwards.

(2) Obturator Nerve

59. **Obturator Nerve.**—This nerve can be stimulated at its exit from the obturator foramen by applying an electrode perpendicularly to the horizontal ramus of the pubis.

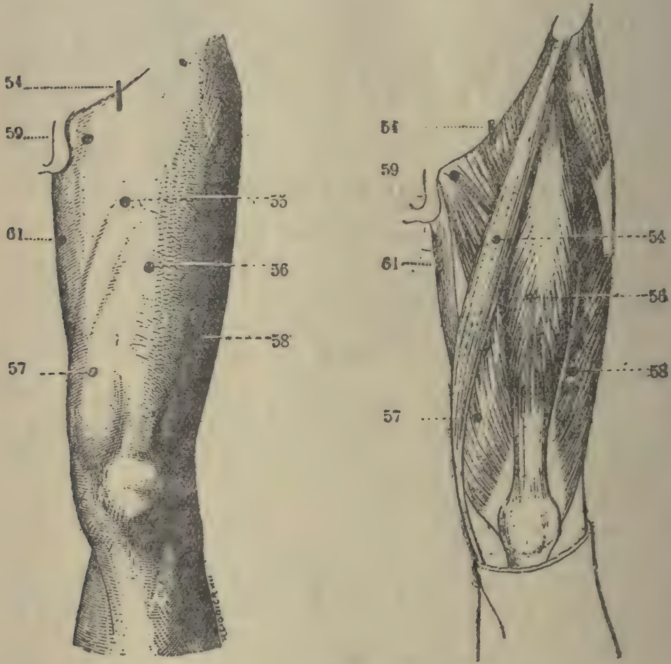
Stimulation causes adduction of the thigh.

60. Adductor magnus.—*Motor point :* in the upper third of the internal aspect of the thigh behind the median line.

Causes adduction and inward rotation of the thigh.

61. Gracilis.—*Motor point :* on the internal surface of the thigh on the median line towards its upper third.

Flexes the leg.



Figs. 29 and 30.—Motor points of the thigh (anterior aspect).

(3) Sciatic Nerve

62. **Sciatic Nerve.**—Point for stimulation: below the gluteal fold, slightly nearer to the tuber ischii than to the great trochanter. Stimulation is difficult in fat subjects.

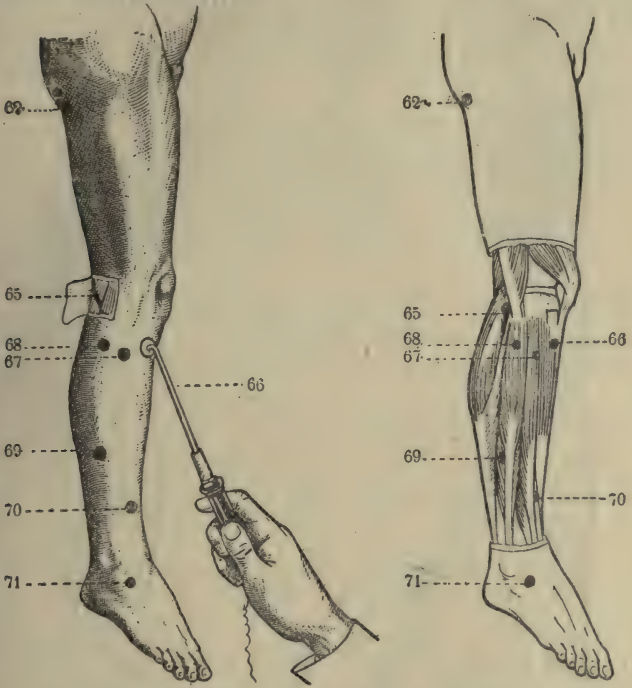
Causes flexion of the leg on the thigh and extension of the foot.

63. Biceps femoris.—*Motor point :* in the middle of the posterior aspect of the thigh, slightly lateral to the median line.

Only a twitch of the muscle can usually be produced.

64. Semi-membranosus.—*Motor point:* towards the middle of the thigh, slightly to the inner side of the preceding.

Only a twitch is produced.



Figs. 31 and 32.—Motor points of the leg (antero-lateral region).

(4) External Popliteal Nerve

65. **External Popliteal Nerve.**—This nerve can very readily be excited behind the head of the fibula as it winds round the neck of that bone.

Stimulation causes flexion of the foot on the leg.

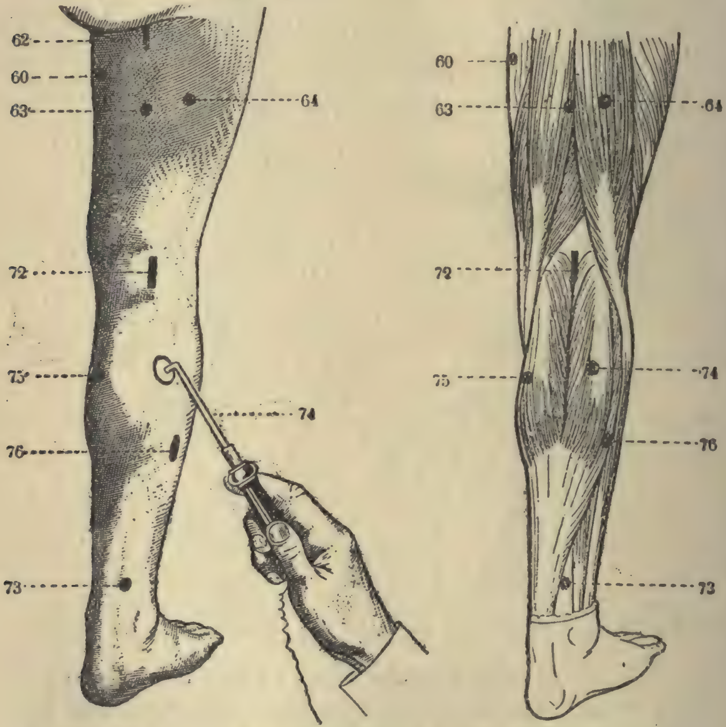
66. Tibialis anticus.—*Motor point:* immediately lateral to the crest of the tibia, 2, 3, or 4 cm. below the proximal extremity of that bone. In fig. 31 the electrode is on this point.

Flexes the foot and elevates its internal border.

67. Extensor digitorum longus.—*Motor point:* lateral and slightly distal to that of the preceding muscle.

Flexes the foot and extends the first phalanges of the toes.

68. Peroneus longus.—*Motor point:* below the head of the fibula, and consequently lateral to the foregoing, but almost in the same horizontal line, or slightly above it. (It should be remembered that stimulation of this muscle lateral to its motor point often gives rise to inversion of the formula.)



Figs. 33 and 34.—Motor points of lower limb (posterior aspect).

Draws the internal border of the foot downwards and the plantar surface of the foot outwards.

69. Peroneus brevis.—*Motor point:* at the junction of the middle and lower third of the leg, in a vertical line through the preceding point.

Causes slight abduction of the foot.

70. EXTENSOR PROPRIUS HALLUCIS.—*Motor point:* between the tendons of the extensor communis and tibialis anticus, close to the crest of the tibia, in the lower third of the leg.

Extends the first phalanx of the great toe.

71. EXTENSOR BREVIS DIGITORUM.—*Motor point:* below and in front of the external malleolus.

Extends the toes.

(5) Internal Popliteal Nerve

72. **Internal Popliteal Nerve.**—*Point for stimulation:* towards the middle of the popliteal space, slightly lateral to the middle line and below the flexion fold of the skin.

Stimulation causes extension of the foot on the leg and flexion of the toes.

73. **Posterior Tibial Nerve.**—The point for this nerve is behind the instep, on the inner side, and the tendo achillis, where the plantar muscles may be stimulated. For convenience, the leg should be flexed, and the patient should lie with the lateral surface of the foot flat on the couch.

74. GASTROCNEMIUS, INNER HEAD.—*Motor point:* in the upper part of the calf, towards the internal border of the muscle.

Extends the foot on the leg.

75. GASTROCNEMIUS, OUTER HEAD.—*Motor point:* in the upper part of the calf, towards the external border of the muscle.

Same action.

76. SOLEUS.—Sometimes *accessible* on the outer and inner side of the leg, where the muscle emerges from beneath the gastrocnemius.

Extends the foot.

FLEXOR LONGUS HALLUCIS.—*Motor point:* immediately lateral to the tendo achillis, 5 or 6 cm. above the external malleolus.

FLEXOR LONGUS DIGITORUM.—*Motor point:* on the internal border of the tibia, adjacent to the internal border of the tendo achillis.

CHAPTER II

ABNORMAL ELECTRICAL REACTIONS

1. Elementary Abnormal Reactions

In a number of affections of the central and peripheral nervous system, the electrical reactions vary more or less from the normal. The study of these pathological modifications of the electrical reactions being a complex one, we shall deal in the first place with the various elementary modifications which may be encountered. We will first examine the way in which these are grouped in different pathological conditions.

These elementary modifications may be quantitative or qualitative.

1. **Quantitative Modifications.**—Evidence of diminution in excitability of a nerve or muscle by the faradic current is afforded by an appreciable increase in the strength of current required for a threshold stimulus. If, for instance, the faradic coil has to be moved up to Division 6 to give the threshold in an affected muscle, while the liminal stimulus for a normal muscle, with the same apparatus, is usually given at Division 10, diminution in faradic excitability, or

faradic hypo-excitability, may be deduced. On the other hand, if in another case an approximation of the coils corresponding to the minimal contraction in a healthy subject, causes relatively energetic contraction of a muscle, increase in excitability will be deduced, or *faradic hyper-excitability* of this muscle.

Diminution of excitability sometimes takes the form, not of requiring a stronger current in order to give the threshold, but of a reduced amplitude of the maximal contraction, as compared with a healthy muscle.

In some circumstances the diminution of excitability is such that with maximum approximation of the secondary coil, the muscle examined gives no response whatever to faradic stimulation. Contraction may then sometimes be produced by substituting a secondary coil giving a current of higher tension (finer winding). If this is ineffectual, bipolar stimulation should be tried. If, however, stimulation by the bipolar method, with the strongest high tension current which the patient can endure, causes no contraction, it may be concluded that the muscle is inexcitable by the faradic current, and that faradic excitability is abolished : this phenomenon is sometimes known as *Duchenne's reaction.*

Dubois (of Berne) has very justly observed that muscles which have been regarded as inexcitable by the strongest tetanising faradic current sometimes respond to *single induction shocks.* This peculiarity should be remembered, for although it indicates a great diminution of excitability, it shows that the latter is not entirely absent.

With the galvanic current, according as the minimal appreciable contraction is obtained with a stronger or weaker current than in the normal state, *galvanic hypo- or hyper-excitability* of the nerve or muscle concerned will be deduced as in the preceding case.

We shall also deduce *abolition of galvanic excitability* when, in an examination conducted with each pole successively, the gradual increase of the intensity of the current within the limits of endurance gives no semblance of a contraction.

Causes of Error.—(1) Hyper-excitability may be merely *apparent* in thin subjects whose nerves and muscles are immediately beneath the skin, or in patients with such anomalies in the situation of the nerves as unusual superficiality, which renders them more accessible to stimuli.

(2) Hyper-excitability and hypo-excitability of nerves and muscles may be merely apparent, in that quantitative variations from the normal are due, not to a modification of excitability, but to *an unusually high or low degree of resistance* in the skin.

In forming conclusions as to alterations in faradic and galvanic excitability, the condition of the skin should therefore be taken into account, since the skin acts as an additional resistance in the way of the current. Variations in its conductivity cause variations in the threshold. Now, as trophic cutaneous disorders are included in the manifestations of many affections of the central and peripheral nervous system, and since these disorders alter the resistance of the skin, it will be understood that this accounts for endless erroneous conclusions. Thus a very resistant skin may conceal increased excitability, and similarly, the coexistence of a slight hyper-excitability, with a somewhat diminished skin resistance, will simulate very pronounced hyper-excitability.

Amongst the causes which reduce skin resistance and are liable to suggest increased excitability, we should mention thinning of the stratum corneum, a moist condition of the skin, a reduction in the subcutaneous layer of fat, and all other causes by which the distance between the nerves and muscles and the electrode may be reduced. On the other hand, scleroderma, hyperkeratosis, dryness of the tissues, vaso-motor disorders, and a lowered temperature, increase the resistance ; similarly, lubricants used for massage (talc or fat), which, finding their way into the glandular ducts, produce similar effects.

These alterations in the conductivity of the skin render the detection of slight modifications of excitability a matter of great delicacy.

2. **Qualitative Modifications.**—The following may be encountered :

(1) Inversion of the normal formula.
(2) Slowness of contraction.
(3) Galvanotonic contraction.
(4) Myotonic contraction.
(5) The longitudinal reaction.

Inversion of the Formula.—In the normal state, in stimulation of a nerve or muscle with the galvanic current, the production of the anodal contraction (ACC) always requires a stronger current than that necessary for producing the kathodal contraction (KCC).

In the pathological state, this relation may be modified, and the anodal contraction may be obtained with an equal or smaller number of milliamperes than that required for producing the kathodal contraction. We say, then, that there is *polar equality* (KCC = ACC) if the two contractions are first appreciable with the same number of milliamperes, and *inversion of the normal formula* (ACC > KCC), if ACC is produced with a weaker current than KCC.

The same modifications, polar equality and inversion, may appear in the relations of the opening contractions; that is to say, the formula may become AOC = KOC or KOC > AOC, in contradistinction from what is found in the normal state. In practice, however, the opening contractions are only rarely investigated, since the changes in the closing contractions usually indicate the elementary reaction of inversion with sufficient distinctness.

This *reaction of inversion* is also sometimes described as *Erb's reaction*, after the author who was the first to study it completely.

In a precise and detailed examination, the degrees of intensity corresponding respectively to the minimal contractions KCC and ACC should always be measured.

Care should be taken to note the degree of intensity determined for each muscle, thus furnishing, for the purposes of later examinations, a record useful as a basis for comparison, and to decide from this whether the inversion is becoming more pronounced, or whether, on the other hand, the anomaly is tending to disappear.

CAUSES OF ERROR.—(1) It should not rashly be concluded that polar equality or inversion is present when these anomalies are observed with a current of any indefinite intensity. The experiments of Chauveau and Boudet, which we have already described, show that in animals, with increasingly strong currents, the contraction ACC becomes first equal to, and finally greater than, the KCC. This phenomenon can scarcely be demonstrated in man in the normal condition, on account of the difficulty in using currents of sufficient strength. On the other hand, this particular inversion is not infrequently observed in the pathological state. It is distinguishable from true inversion in that, contrary to the latter, it is not produced at the threshold. Whereas *in true inversion the AC contraction is given when the KC stimulus gives no response*, in inversion associated with Chauveau's phenomenon, it is merely that the *force* of the AC contraction is greater than that of the KC contraction.

Inversion at the threshold is also relatively less common, and we believe that it has a much more definite and important significance. The inversion associated with Chauveau's phenomenon may indicate a pathological condition, or a variation of excitability, but inversion at the threshold is evidence of serious and far-reaching disorder in the structure of the muscle.

(2) Inversion is usually associated with slowing of the contraction. It may, however, be observed in the normal state, with lively contraction, in perfectly healthy muscles. Those most frequently presenting this anomaly are: supinator longus, the deltoid, the inner head of triceps, the abductor digiti quinti, and first interosseous, in the upper limb; the gluteals, the vastus internus, peroneus longus, and the gastrocnemius, in the lower limb.

Inversion of the formula in these cases results from incorrect placing of the testing electrode, its application outside the point of election, or the use of an electrode of too large dimensions relative to the muscle tested. A slight alteration in its position, by 1 cm., for instance, is often sufficient to reproduce the normal reaction.

This false inversion is easily explained by reference to Pflüger's law of the origin of stimulation.

With the electrode in a wrong position, the KC stimulus falls on a point of lower excitability than the motor point. At anodal closure, however, the stimulus from the indifferent pole arrives at the extremity of the muscle, and there creates a virtual pole which may be of high density, and may act on a more excitable region than that over which the electrode is placed.

(3) If the intensity of the threshold of KCC is noted in milliamperes, and that of the threshold of ACC immediately afterwards, equal values may be obtained, or the ACC may even be given with a lower intensity. Here again polar equality or inversion may be only apparent. The passage of the current giving the KCC, while the instrument is being read, lowers the resistance of the organism. The AC stimulus following the KC is therefore passing through a less resistant circuit, and will consequently require an equal or smaller number of milliamperes.

The anomaly disappears if the values are read only after a number of closures with each pole alternately.

(4) Examinations carried out without the help of a double key may also reveal an apparent inversion.

The passage of the current at the moment of kathodal closing produces a polarisation current in the tissues which is antagonistic to the current producing it. If this be followed rapidly by an anodal closing stimulus, the strength of the latter is increased by that of the polarisation current, and the AC contraction, produced by a current of greater intensity, becomes equal to, or more forcible than, the KC contraction.

This source of error is obviated by the use of a double key. The polarisation current is short-circuited though this instrument at the moment of release of either key (see p. 9).

Slowing of the Contraction.—It is essential in electro-diagnosis to observe the nature of a muscular response.

The contraction, which is normally short, sharp, and of lightning rapidity, may undergo changes in form, more commonly becoming *slowed*, or definitely *slow, sluggish, and vermicular.* A more or less diffuse wave in the muscle can be observed beneath the skin, and the contraction suggests somewhat the creeping movement of an earthworm.

This anomaly was discovered by Remak.

The slowness of the contraction is sometimes difficult to detect, particularly when it is not pronounced: in such difficult cases, an indisputably healthy muscle in the same subject should be used as a means of comparison.

The slowness is almost always *more marked* in the AC contraction than in the KC. In cases where it is slight, it may even be completely absent in KCC, which remains characteristically lively, and only be evident in ACC.

In other cases, slow contractions become progressively more rapid as the examination proceeds. Such phenomena are associated with partial reaction of degeneration.

Slowness of contraction is not peculiar to galvanic excitation. It is often similarly observed in faradic excitation, when excitability to this form of current is retained.

Analysis of the slow contraction by means of the graphic method, and comparison with a normal tracing, show that slowing occurs in all the different stages of the contraction; the latent period is lengthened, the upstroke of the shortening period is less abrupt, and the lever remains at this height for some time, whereas a normal myographic curve exhibits a sharp apex (fig. 36). Lastly, the period of relaxation is prolonged, particularly towards the end. It should be noted that it is always in this last stage, that of relaxation, that slowing is most pronounced and characteristic.

Larat's suggestion that the nature of a contraction should always be recorded in this way, is certainly attractive, since it would afford permanent records, and facilitate comparisons of great value in determining the evolution of muscular changes. Unfortunately an electro-diagnostic examination is already a lengthy process, and it is hardly to be expected that electrologists will acquiesce in a complication in technique when its purpose can always be served by a careful examination.

CAUSES OF ERROR.—Sources of error in estimating rapidity of contraction will be studied in connection with the reaction of degeneration (see p. 77).

Galvanotonic Contraction.—We have seen that, in the normal state, a relatively strong current applied to a nerve causes at kathodal closure, either a single forcible contraction or a prolonged or "tetanic" contraction, which is, more correctly, called tonic. This continues while the current is passing, and ceases at the break.

This galvanotonic contraction is easily obtained by stimulation of the nerve, but almost impossible to produce over the muscle, in the normal state. But in the pathological condition it may be obtained with relatively low intensities. Erb, who first discovered this, observed that, in the reaction of degeneration, the slow contraction sometimes assumed the character of a tetanus, and continued until the circuit was broken. Since then it has frequently been observed by Huet, Delherm, Bourguignon, and by the authors.

It does not usually occur when the muscle is stimulated indirectly through the nerve, when the latter retains its excitability, but on direct stimulation of the muscle at the motor point. Sometimes it characterises the entire contraction, which is tonic from the outset. Sometimes it follows a slowly initiated contraction, which is thus succeeded, not by complete relaxation, but by a continuous state of contraction, relaxation only occurring when the circuit is broken.

In general it is more obvious with the *anodal* than with the kathodal stimulus.

It is most readily detected by stimulation *in the neighbourhood of the tendon* (longitudinal excitation), rather than at the motor point (the kathodal electrode being over the tendon).

The peculiar feature of this galvanotonic contraction is that its duration coincides with that of the current. *It ceases when the circuit is broken.* For this reason it has been called temporary galvanotonic contraction, in distinction from permanent galvanotonic contraction, which persists after cessation of the stimulus, an important characteristic of the myotonic reaction (Thomsen's disease).

Permanent Galvanotonic Contraction differs from the preceding in that it persists after the circuit has been broken. It is produced by sufficiently strong currents with either pole at the motor point. It is most definite in AC, or better still, with longitudinal stimulation. It persists for several seconds after cessation of the stimulus, before relaxation occurs. It may disappear with repeated stimuli, which reduce its duration. It forms part of the myotonic reaction, and occurs in certain muscles at the onset of myopathies.

The Longitudinal Reaction.—In the normal state, muscles are excitable anywhere in their fleshy portion, but their excitability reaches an optimum in the small area of the motor point. Variations in excitability which occur in the muscle in a pathological condition, and become evident on stimulation of the motor point by the unipolar method, are not confined to this point, but affect the entire muscle. Thus the distal extremity of an affected muscle responds more readily than in the normal condition. Whereas a strong galvanic current is required for producing contraction in a healthy muscle by stimulation at the junction with its tendon, a relatively weak current may suffice for this if disease is present. When at the same time excitability at the motor point is markedly reduced, or even absent, stimulation at the junction with the tendon will be found more effective, and may even entirely replace excitability at the motor point.

Discovered by Remak (1876), again by Doumer in 1891, and studied shortly afterwards by Huet (1894), this phenomenon was described by Wertheim-Salomonson as displacement of the motor point.

Later, Ghilarducci, having shown that it could be obtained by stimulation below the muscle, even, indeed, at the extremity of the limb, named it the remote reaction of degeneration, an expression which was used for some time, but is to-day almost universally replaced by the terms longitudinal hyper-excitability, or longitudinal reaction, the name given to it by Huet.

Longitudinal reaction or hyper-excitability, displacement of the motor point, and remote reaction of degeneration are therefore different names for the same phenomenon. In symbols it is often written RDd.

It has long been regarded as a late or terminal manifestation of the reaction of degeneration, but it is now well established that it accompanies the latter throughout its development, and constitutes one of the earliest and most important elementary reactions (sometimes appearing two or three days after injury to the nerve).

When the testing electrode is applied to the lower extremity of the belly of a muscle which is degenerating, at a short interval after the commencement of this process (fig. 35), it is observed that the closing stimulus at this point causes contraction with a much weaker current than would be effective in the normal state. Longitudinal hyper-excitability is present; and this hyper-excitability is actual, and *absolute*. Later, excitability diminishes at the motor point, and diminishes equally to longitudinal stimulation, a stronger current being required than in the normal state. But the diminution in excitability being less to longitudinal stimulation than at the motor point, longitudinal stimulation continues more effective than stimulation at the motor point; hence there is *relative* longitudinal hyper-excitability.

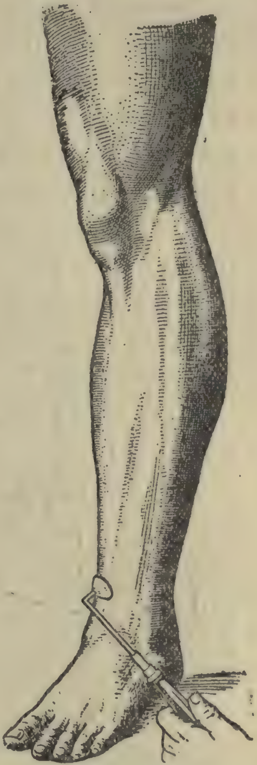


FIG. 35.—Examination of the longitudinal reaction on the tibialis articus muscle.

When excitability at the motor point is abolished, excitability at the point of junction with the tendon may be the only evidence of excitability in the muscle.

The contraction following longitudinal stimulation is a manifestation of degeneration, and possesses the essential characteristic of degeneration phenomena, which is *slowness*. This feature is often even more pronounced on longitudinal stimulation than at the point of election. The contraction produced by stimulation of the motor point may even seem lively, or its nature may appear doubtful, but its sluggish nature will usually be obvious on stimulation in the region of the tendon.

The order of appearance of contractions in longitudinal stimulation does not differ from the normal order, at least in the majority of cases; KCC is obtained as a rule before ACC, but inversion occasionally occurs.

In the initial phases only, of the reaction of degeneration, a galvanotonic, instead of a slow, contraction, is sometimes obtained. The other reactions of the muscle are usually perfectly normal at this stage; stimulation at the motor point gives no slowed contraction, and shows no modification of the formula. It follows that the independent occurrence of a tonic contraction in longitudinal examination may be a very early and delicate sign of a change in the muscle (Bourguignon).

Rich's Reaction, or the Reaction of Compression.—The reaction of Rich, or, more exactly, of Richard Geigel, is a quantitative reaction following compression of the nerves and vessels with the fingers, or with Esmarch's bandage. It is characterised by the early appearance of the opening contractions on stimulation of the nerve.

Supposing that in the normal state the succession of contractions is obtained with the following intensities:

KCC	1 ma.
ACC	2 ma.

AOC	3 ma.
KOC	10 ma.

After compression with the fingers or Esmarch's bandage they are obtained as follows :

KCC	1 ma.
ACC	1 ma.
AOC	2 ma.
KOC	2½ ma.

It will be seen from this that the intensities required for obtaining the opening contractions, particularly KOC, are very low in comparison with the normal.

The explanation of the phenomenon probably lies in the fact of existence of a polarisation current, which is superimposed on the principal current at the break, and is of unusual strength because the products of electrolysis, which are the cause of polarisation, cannot be carried away by the blood-stream on account of the compression.

The phenomenon becomes more marked the longer the current is kept closed, which would appear to verify this theory.

II. Grouping of the Elementary Reactions

In nervous pathology, when the responses of nerves and muscles to electrical stimulation vary from the normal, the anomaly may consist solely in an increase or reduction of excitability ; such a modification is purely quantitative. In other cases the anomaly is characterised both by quantitative modifications and by a change in the form of the muscular contraction, which may or may not be associated with an alteration of the normal order of appearance of the contractions.

The reaction of degeneration falls in the last category.

Normal Reactions.—Normal excitability is preserved (or, to be less dogmatic, only varies slightly) in the following:

1. Affections, slight or even serious, of the brain and spinal cord (epilepsy, tabes, chorea, paralysis agitans, cerebral concussion, etc., etc.).

2. Affections of the peripheral motor neurone, liable to give rise to the reaction of degeneration, but failing to do so owing to the mild nature of the morbid process.

3. Affections of the neuro-muscular system of a functional nature. The motor paralyses of hysteria show no modifications in the electrical reactions. Sometimes, however, slight hypo-excitability is observed in hysterical subjects. In certain cases this is merely apparent, resulting from the increase in skin resistance which frequently occurs in hysteria; in other cases the muscles are found to be in a spastic condition, and it is supposed that a stronger current is required to give the threshold in spastic muscles than in a normal subject, whose muscles are completely relaxed.

III. Increased Excitability

Increased excitability is characterised by the appearance of the threshold with weaker currents than in the normal state.

If only slight it is not easily detected, and its existence may remain uncertain. In bilateral affections, the only means of comparison is with the usual values of the faradic and galvanic threshold in the healthy subject, but in the case of a unilateral lesion, there is no possibility of doubt unless the differences in excitability between the two sides are only extremely slight.

An increase in excitability occurs in recent organic cerebral paralyses, particularly if these are accompanied by spasticity; in certain forms of myelitis, in the first stage of tabes, disseminate sclerosis, Little's syndrome, tetany, writer's cramp, reflex contractures, and in a general way in the majority of affections in which the tendon reflexes are exaggerated.

IV. Reduced Excitability

Simple diminution of excitability is commoner than augmentation. It is recognised when the threshold stimulus, with both forms of current, requires a greater strength of current than in the normal state.

As with hyper-excitability, slight degrees of it are sometimes difficult to detect.

Sometimes it is more marked with one of the forms of current than with the other, either with the faradic, or with the galvanic.

Lastly, it is often more marked on stimulation of nerves than of the muscles.

In certain cases the diminution in excitability of the nerves is such that with ordinary electro-diagnosis apparatus, using the bipolar method, no response whatever can be obtained; excitability may then be considered abolished.

Sometimes, in severe reflex atrophies or myopathies, for instance, the loss of contractile substance in the muscle may be so extensive that the muscle will not respond to electrical excitation: in such a case the excitability of the muscle is abolished.

Simple hypo-excitability is observed in nerves and muscles in old cerebral paralysis and in anterior poliomyelitis, but then only in muscles slightly affected, and capable of rapid regeneration; in hæmatomyelitis, reflex muscular atrophies or atrophy due to functional inactivity; in therapeutic immobilisation, the voluntary immobilisation of malingerers, or the unconscious immobilisation of hysterical subjects, but in these cases it exists only in a slight degree: it is always more pronounced when it corresponds to an atrophy following a fracture or articular lesions. It is the rule in myopathies, in certain slight peripheral paralyses, benign facial palsy *a frigore*, the majority of peripheral paralyses by slight temporary compression, etc.

Primary myopathies are accompanied by a hypo-excitability which increases in degree as the atrophy becomes more advanced. In the early stages, when the amyotrophia is still only slightly perceptible, the diminution in excitability takes the form, not so much of a raised threshold as of a reduced amplitude of contraction (Huet).

In infective or toxic neuritis (alcoholic and diphtheritic paralyses, etc.), simple hypo-excitability justifies a favourable prognosis.

Hypo-excitability also appears in traumatic peripheral neuritis, infective or toxic neuritis, at a late stage of their development, following after the reaction of degeneration. At this period the power of voluntary movement may have been wholly, or only partially, recovered.

In cases which have not been cured, *i.e.* in which the power of voluntary movement has not been regained, excitability is always greatly diminished. Usually only traces of it persist.

Cases of Extreme Hypo-excitability. — Very profound hypo-excitability, sometimes even so profound that no faradic response is obtained, and no galvanic current of less than 20, 25, or 30 milliamperes will excite contraction, is sometimes met with in certain acute processes with severe clinical symptoms, such as cerebro-spinal meningitis, poliomyelitis, certain forms of toxic or infective neuritis, traumatic neuroses, and nerve compressions (Huet).

Sometimes the whole area affected shows this hypo-excitability; at other times muscles or other nerves in the immediate neighbourhood definitely show the reaction of degeneration.

A peculiarity of these cases is that, in spite of the severity of clinical signs which suggest the existence of RD (severe compression, for instance), nothing is found beyond hypo-excitability. It is then discovered that with this marked hypo-excitability, bordering on inexcitability, and often characterised by complete loss of faradic excitability, the galvanic contraction, which might well be expected to be slowed, is still brisk.

It might also be supposed that this hypo-excitability represents the last stage in the course of a reaction of degeneration, not previously recognised, owing to its rapid development, but this is by no means the case, since excitability diminishes *very rapidly from the onset of the affection.*

However this may be, experience shows that in these cases where serious clinical signs are associated with a hypo-excitability of disquieting severity, *the prognosis is relatively favourable*, and recovery is readily brought about.

When, in a region showing RD are found muscles exhibiting this symptom of pronounced hypo-excitability, it is invariably in the latter that the power of voluntary movement is regained more rapidly and completely.

According to Huet, marked hypo-excitability is commonest in the lower limb, in the muscles of the thigh, more particularly the biceps and vastus internus, and in the posterior muscles of the leg, particularly the gastrocnemius.

V. The Reaction of Degeneration

By the reaction of degeneration (RD) is understood a syndrome composed of the following elementary electrical reactions:

1. The excitability of the nerve (faradic and galvanic) is abolished.

2. The faradic excitability of the muscle is abolished.

3. The galvanic excitability of the muscle is either increased or diminished.

4. The following qualitative changes, on galvanic stimulation of the muscle, are present:

(*a*) Slow or galvanotonic contraction.

(*b*) Inversion of the formula (ACC > KCC), or polar equality (ACC = KCC), or the normal formula KCC > ACC.

(*c*) Longitudinal hyper-excitability.

When these modifications are coexistent and definite, *the reaction of degeneration* is said to be *complete.*

When a motor nerve in an animal is divided, changes in the electrical reactions of the nerve and the muscle appear, usually on the second day, develop for several weeks, and at a certain stage in this development give the picture of complete RD. Hence, certain authors have proposed to distinguish complete RD by the term "syndrome of nerve section."

The expression "reaction of degeneration" originates in the fact that this electrical syndrome appears when a nerve undergoes Wallerian degeneration.

This parallelism is not, however, admitted by all authors. Strumpell, for instance, was unable to detect any degenerative lesion, apart from proliferation of the nuclei, in excised muscles showing all the characters of RD, and disputes the degenerative significance attributed to the reaction of degeneration, explaining it simply as a reaction on the part of the muscle itself when deprived of its nerve supply.

Similarly, Babinski, taking account of the fact that the characters of RD may appear extremely early, at a period when the muscle cannot have undergone morphological change, three or four days after section of the facial nerve, for instance, regards the reaction of degeneration as the response of a muscle released from the control of the nervous system.[1]

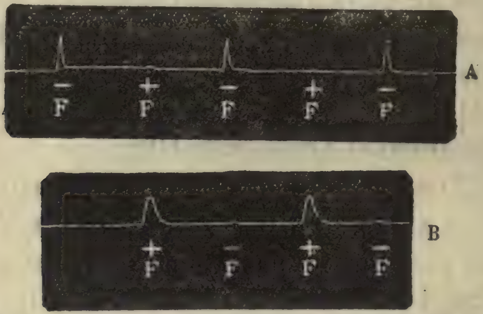


Fig. 36.—Diagrammatic representation of muscular responses to galvanic stimulation (closing contractions).

(A) Normal muscle.—At $\overset{-}{F}$ (kathodal closure) the contraction is brisk, and more forcible than at F (anodal closure).

(B) Muscle showing the reaction of degeneration (with inversion of the formula).

At $\overset{+}{F}$, anodal closure gives a slow contraction, more forcible than that produced at $\overset{-}{F}$ (kathodal closure).

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[1] Pitres has shown that the slowing of the muscular contraction obtained by percussion, in peripheral nerve lesions, coincides with disappearance of faradic excitability in the muscles. He therefore advised clinicians with no electro-diagnosis apparatus at their disposal to resort to percussion as a substitute, regarding slow contraction on percussion as adequate evidence of RD. That this is a mistake is clearly shown by the slowed contraction obtained on percussion in reflex paralysis (Babinski, Hallion, and Froment, "Slowed muscular contraction obtained by percussion, and its clinical significance, studied by the graphic method," *Soc. Neurol.*, June 29th, 1916).

Partial Reaction of Degeneration.—The reaction of degeneration does not always appear in the form which we have just described. Frequently the quantitative modifications exist only in a slighter degree; the nerve has not entirely lost its excitability, but only shows diminution in faradic and galvanic excitability to a greater or less extent; similarly, the muscle may still respond to more or less vigorous faradic stimuli. The other features of RD are, however, identical.

In brief, partial reaction of degeneration is constituted by the following elementary reactions:

1. The excitability of the nerve (faradic and galvanic) is more or less diminished.

2. The faradic excitability of the muscle is more or less diminished.

3. The galvanic excitability of the muscle is either increased or diminished.

4. The following qualitative changes appear in the muscle on galvanic stimulation:

(*a*) Slow or galvanotonic contraction.

(*b*) Inversion of the formula ($ACC > KCC$), polar equality ($ACC = KCC$), or the normal formula ($KCC > ACC$).

(*c*) Longitudinal hyper-excitability.

In certain cases the diminution in faradic excitability may be very slight or even imperceptible, the KC contraction may remain brisk, and slowing only appear in ACC, or even only on longitudinal excitation. When this milder combination is present, it is said that there are *traces of the reaction of degeneration.*

Symptomatic Value of the Slow Contraction and Inversion.—Complete and partial reaction of degeneration consist, therefore, in an association of certain elementary reactions, and not, as is still sometimes said, merely in inversion of the galvanic formula. Of all the elementary reactions which together constitute the syndrome of degeneration, inversion of the formula is actually *the least constant*, always provided that it is tested for *with liminal stimuli*, and not with stronger currents. At the threshold, inversion is relatively uncommon.

The contrary applies to *the slow contraction, or its equivalent, galvanotonic contraction, which is in some sense pathognomonic of RD.* If slowed contraction or galvanotonic contraction is absent, RD is not present.

In one variety of the condition, it is true, slowing is absent, but this absence is only apparent. This occurs when, as in certain chronic affections in which the degenerative process, instead of attacking the peripheral motor neurone as a whole, gradually affects one motor cell after another, or one nerve fibre after another, the reactions of the altered muscle fibres are masked by those of the fibres which are still healthy. In this way the slow contraction is concealed by the brisk contraction of the unaffected fasciculi, and only a more or less marked simple hypo-excitability is detected. Later, when the fasciculi originally immune are affected in their turn, the fasciculi originally attacked have become almost inexcitable ; hence there is marked hypo-excitability of the whole muscle, which militates against satisfactory detection of the qualitative changes characterising RD (Huet). It should be added that slowed contraction can be observed in cases of this kind in superficially situated muscles, a contraction which, however, is briskly initiated, a fact for which the coexistence of healthy and degenerated fibres is an obvious explanation.

The slow contraction is usually observed only on direct stimulation of the muscle. Thus in partial RD, in which the nerve is still excitable, direct galvanic stimulation of the muscle gives the slow contraction, whereas stimulation of the nerve (galvanic or faradic) gives a brisk contraction, and no polar inversion.

We shall see later, however, that slowing may sometimes be shown on excitation of the nerve (Remak's faradic reaction of degeneration).

CAUSES OF ERROR.—There can be no doubt,—and this point has recently been strongly emphasised,—that slowness of the contraction may exist apart from the reaction of degeneration.

Experimentally, in the frog, modifications in the form of the normal contraction have been obtained by the use of various substances—caffeine, quinine, veratrine, and salts of potassium and ammonium. Cluzet observed slowing of the contraction after the injection of spartein.

Curare acts in the same way, causing temporary paralysis of the motor nerve fibres; the muscle becomes almost insensible to induction shocks (Brücke); its galvanic excitability remains almost normal, but the contraction is slowed (Cluzet). When the effect of curare passes off, the electrical reactions become normal.

Clinically, in man, in certain general affections associated with cachexia, such as diabetes, jaundice,[1] the cachexia of new growth, and tuberculosis,[2] a certain *prolongation of the contraction* is sometimes seen.

Further, exhaustion from excessive faradic stimulation (Dubois), CO_2 poisoning, and cold, may cause slowing.

The effect of cooling on muscular contraction in the frog and other cold-blooded animals was recognised long ago by Helmholz, Pflüger, Marey, and others. In warm-blooded animals, prolongation of the contraction following a reduction of temperature appears, on the contrary, to have been less closely studied. Bernhardt, however, and Hitzig, have drawn attention to a prolongation of the faradic contraction, and others have subsequently demonstrated clearly that a contraction, slowed by cold, recovers its rapidity when the muscle is warmed.[3]

Grund[4] was the first to speak of a "reaction of cooling," and to attempt to distinguish it from the reaction of degeneration.

Since faradic excitability is preserved, the reaction of cooling might simulate partial reaction of degeneration, and this would explain how authors have been able to discover RD in affections in which it does not occur. In this way Bernhardt imagined that he had detected RD in a case of Raynaud's disease.

The pseudo reaction of degeneration *a frigore* is sometimes associated with polar equality, or with inversion. Sometimes the slowness of the contraction is as marked as in the most pronounced RD. Prolongation of the contraction is particularly evident in slender muscles, more especially the small muscles of the extremities. But with a considerable lowering of temperature, the phenomenon may appear in the larger muscles.

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[1] Edinger, *Zeitschr. f. Klin. Med.*, 1883.

[2] Reiss, *Die elektr. Entartungsreaction*, Berlin, 1911.

[3] Graphic records show that there is no increase in the latent period, but a prolonged phase of contraction, an interval, and slow relaxation.

[4] Grund, *Zentralblatt f. Neur.*, 1908, et *Deuts. Zeits f. Nervenheilkunde*, 1908, p. 172.

According to Reinecke,[1] excitability shows some slight modifications; it is slightly increased in both muscle and nerve to the galvanic and faradic tetanising currents, but diminished to single shocks. These changes are more evident in stimulation of the nerve.

The distinguishing point from partial RD is the immediate disappearance of the slowing on warming or exercise.

A slowed contraction may be produced, not only by the action of external cold, but by *hypothermia* of the limb from vaso-motor disturbance. It seems probable that the phenomenon results from asphyxia of the muscle by the accumulation of CO_2.

In reflex paralyses, slowed contraction appears when vaso-motor disturbance is present; rapidity of contraction is decidedly regained, and may even become normal on the application of heat or by removal of the exciting cause.

The cooling of a dead body acts in the same way. Babinski, on the one hand, and Marie and Cluzet, observed slowed contraction, with polar inversion and faradic inexcitability, one hour after death. Cut off from indirect stimulation through the motor nerve, the death of the latter being is a more rapid process, the muscle would thus react in accordance with its own properties of excitability (Babinski).

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[1] Reinecke, *Zeitsch. f. allg. phys.*, 1908, p. 422.

External cold and lack of oxygen may also affect faradic excitability in the limbs. Faradic slowing, however, is not always as obvious to the eye as galvanic slowing, but it may be detected by determining the number of stimuli (per second) required to produce tetanus. The number is invariably lower than the normal, *i.e.* there is *premature fusion of the contractions.* In practice this effect gives rise to great difficulty when a patient for examination, with a peripheral paralysis, is also suffering from vaso-motor disorder and hypothermia.

Similarly, these effects should be taken into consideration during very cold weather, as we had reason to know during the severe winter of 1917. Numerous mistakes in electro-diagnosis were made. Reflex paralyses, or even psychic paralyses associated with slowed contraction, were described and put down as reaction of degeneration, mistakes in diagnosis which might easily have been avoided, either through observing that the same slow contraction was given in regions where there was no question of a nerve lesion, or by adequate warming of the limb examined.

From the fact that cold may cause slowing of the contraction, it does not follow that this sign should never be regarded as a valid indication of the reaction of degeneration. It is perhaps necessary, in order that it may continue to be regarded as pathognomonic of RD, to specify the conditions in which it is valid; the following words might accordingly be added to its definition: "and at the normal temperature of the body," thereby paraphrasing the words, "and at the same temperature," found in the laws of physics and chemistry.

Intermediate States between the Complete and Partial Reactions of Degeneration. — 1. *The preservation of excitability to single induction shocks.*—The abolition of faradic excitability in the muscle usually refers to the rapidly interrupted faradic current. At the same time it is not uncommon to find absolute inexcitability to this form of faradic current when the muscle still responds fairly well to isolated induction shocks (Dubois[1]). Certainly the strength of the latter must be adequate, and the maximum is usually required for producing any response. The muscle responds to these single induction shocks by a sluggish contraction, not, however, exhibiting the slowness characteristic of galvanic stimulation, but none the less apparent, and readily verified by the reduction of the number of the stimuli necessary to cause tetanus.

This peculiarity of excitability in the muscle should be remembered. It shows that faradic inexcitability cannot be deduced until it has been shown, by the bipolar method, that there is no reaction whatever either to the tetanising current, or to single induction shocks.

2. *The Latent Faradic Reaction.*—Babinski, Del-

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[1] Dubois, *Korrespondenzbl. f. Schw. Aertzte*, 1888.

herm, and Jarcowski[1] have shown that in certain cases of complete RD, the faradic excitability of the muscles may reappear if a continuous galvanic current be passed through the limb, while the faradic reactions are being tested. They have not, however, succeeded in re-establishing faradic excitability in the nerve.

This phenomenon, described by these authors under the name of "latent faradic excitability," indicates a less serious condition of the muscle than does complete abolition of faradic excitability, and should lead to a less unfavourable prognosis than in the case of complete RD.

This return of faradic excitability under the influence of a continuous current appears comparable to the phenomena of resensitisation of a degenerated muscle by certain chemical substances. Salts of sodium and potassium, for instance, and veratrine, which have little action on normal muscular tissue, appear, on the other hand, to exert very profound effects on a muscle which has degenerated. Muscles not previously responding to faradic stimulus become excitable by this form of current under the influence of these bodies, possibly, as Mlle. Joteyko suggests, owing to their selective action on the sarcoplasm, which they render sensitive. Whatever explanation is accepted, it is probable that the passage of the continuous current through the muscle exercises on the latter its electrolytic effects. The chemical substances produced increase its excitability, and thus enable it to respond to the faradic stimulus.

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[1] Babinski, Delherm, and Jarcowski, *Soc. fr. d'Électroth.*, March 1912.

3. *The Faradic Reaction of Degeneration.*—We have shown above that in partial RD, stimulation of the nerve (either faradic or galvanic) usually produces a brisk contraction, whereas direct galvanic stimulation of the muscle provokes a slow contraction.

In certain processes, however, a muscle stimulated with the faradic current, directly on its motor point, or indirectly through the nerve, responds by a more or less slow contraction. Slow contractions with both galvanic and faradic currents are therefore associated. Remak distinguished this combination by the name "faradic reaction of degeneration." If the nerve has lost all faradic excitability, and the muscle has retained it, a slow contraction following faradic stimulation of the muscle is sufficient to characterise the faradic reaction of degeneration.

Nevertheless this reaction has no special diagnostic or prognostic significance, and does not call for separate consideration : it constitutes one of the many guises which partial RD may assume, and merely indicates a more profound change in the muscle than partial RD with a brisk faradic response.

VI. THE EVOLUTION OF THE REACTION OF DEGENERATION

The elementary reactions which together constitute the reaction of degeneration, *characterise its stage of* *maturity*. This period is preceded by one of initial development, of short or long duration, according as the pathological process producing it is acute, subacute, or chronic. It is followed, on the other hand, by a terminal phase, which varies in nature according as the progress of the affection which caused it allows recovery or permanent inexcitability (progressive or regressive changes). The reaction of degeneration, such as we have defined it, is therefore not a fixed, lasting, and definite syndrome, but merely *a transitory and passing manifestation*.

Initial Period.—For some hours following experimental section of a nerve, an increase in both faradic and galvanic excitability is observed in the peripheral end of that nerve. This phase of initial hyper-excitability is very brief, lasting usually not more than about forty-eight hours, and is followed rapidly by a progressive diminution of excitability, which terminates towards the end of the first week in absolute inexcitability. This initial hyper-excitability probably also exists clinically, but since very early examinations are rarely carried out, this occurrence generally escapes observation, and the first condition to be detected is *inexcitability of the nerve*. Meanwhile *the faradic excitability of the muscles diminishes* more or less rapidly during the hours following division of the nerve, and disappears towards the second week.

Galvanic excitability, after a stage of diminution, which is usually very brief, becomes greater than normal, *i.e.* we have galvanic hyper-excitability; and it was precisely this combination of galvanic hyper-excitability and decreased faradic excitability of the muscle that astonished the first observers who obtained these reactions clinically (Baierlacher, Ziemssen, and Erb).

The duration of the phase of galvanic hyper-excitability varies considerably; in certain circumstances it is very short, and is rapidly succeeded by diminution in galvanic excitability; in other cases it is very prolonged, and continues beyond the initial period, hyper-excitability being still evident at the period of established RD.

At this stage of hyper-excitability the opening contractions, particularly KOC, are sometimes obtained more readily than usual (Lugard). This peculiarity recalls what is observed in nerve compression (Richard Geigel's reaction)[1]; but here we are concerned with a reaction proper to the muscle, and not to the nerve. The appearance of opening contractions in a muscle is always, therefore, in view of the difficulty of obtaining them in the normal state, a somewhat suspicious sign. Hyper-excitability to opening stimuli, like galvanic hyper-excitability itself, except in facial paralysis *a frigore*, is merely transitory.

Along with these quantitative modifications, the following qualitative modifications progressively develop: longitudinal hyper-excitability, slowing of the contraction, and anomalies in the order of appearance of the contractions. The latter, however, is a relatively late sign, and never appears before the second week.

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[1] See p. 66.

Stage of Maturity.—This period is defined by the classical syndrome which we have analysed in detail. No further description is necessary.

Progressive Changes.—The progressive changes of RD are concerned solely with the galvanic excitability of the muscle, since both nerve and muscle have permanently lost their faradic excitability, and the nerve its galvanic excitability.

A common phenomenon of secondary importance is a more pronounced slowing of the contraction ; the effect of the positive pole also usually comes to preponderate.

The essential phenomenon is progressive diminution in the galvanic excitability of the muscle. The strength of current required for obtaining the threshold becomes continually greater, and finally, *it may be long after the onset of the affection* (*from several months to one or even two years*), galvanic excitability at the motor point disappears completely.

When excitability at the motor point has been lost, the muscle may still be made to contract by a stimulus applied at a distance from its motor point, and the further period during which it continues to react to longitudinal excitation may also be prolonged for many months.

Loss of excitability at the lower extremity of the muscle does not, however, mean that the muscle has become totally inexcitable. It is, in fact, possible, by *localising* the galvanic stimulus, as in localised faradisation, to obtain evidence of electrical contractility. The extensors of the forearm, for instance, when absolutely inexcitable by the galvanic current with the indifferent electrode applied on the back, will respond with very definite slow contractions, on stimulation, whether remote or at the motor point, if the indifferent electrode be applied over the lower part of the triceps brachialis, above the olecranon.

The successive periods of change in the electrical reactions which have been described, initial period, reaction of degeneration properly so-called, and the phase of DRd only, represent the course of complete RD in its entirety. Such, in fact, is the succession of effects always associated with an anatomical process producing the definite lesions of Wallerian degeneration.

It occurs after division of a nerve, and in all affections in which severance of connection with the trophic centres gives rise to lesions of the same order and the same degree. In other conditions, however, RD does not necessarily pass through all these stages. The process of its development may cease at any time, and recovery commence, if and when the histological changes underlying the condition are arrested.

Regressive Changes.—Regressive evolution is characterised by a tendency on the part of the pathological reactions to approach more nearly to the normal.

In the majority of cases this return of the reactions towards the normal is *preceded for a long or short period by a return of the power of voluntary movement.*

In lead paralysis of the extensors, for instance, the patient may entirely have recovered the use of the limb at a time when electrical examination still reveals the existence of RD.

The first sign of regression is, usually, reappearance of the excitability of the nerve. The nerve begins to react to galvanic stimulation, and then to the faradic current, the reaction to *single induction shocks* being the first to appear. In this way a brisk muscular contraction is produced, whereas faradic excitation of the muscle is still without effect, and the galvanic current gives a slow contraction. The muscle then regains its faradic excitability, at first only partially, and less completely than the nerve, while the galvanic contraction, on the other hand, is gradually losing its characteristic of slowness, and excitability is *moving upwards* towards the region of the motor point. The longitudinal reaction ceases to predominate over the reaction at the motor point, and KCC becomes equal to, and finally greater than, ACC.

This process, which is always a matter of months, invariably indicates anatomical repair.

It should be borne in mind that long after complete clinical recovery, particularly in cases where the affection has been considerably prolonged, traces of the disorder, in the form of a few definite signs, may still be present.

Inversion, for instance, may persist for a long time, either as complete inversion or as polar equality, and signs of longitudinal hyper-excitability may still be found in muscles previously involved, several months or even years after the disappearance of RD. Lastly, quite the commonest sign to persist is *a simple diminution of excitability* in both nerves and muscles, less marked, however, in the nerves, which regain almost their normal degree of excitability, than in the muscles, the excitability of which continues very diminished for a considerable length of time, more particularly for the galvanic current.

Acute, Subacute, and Chronic Development of RD.—In certain affections, RD passes rapidly through its different phases, and arrives at the terminal stage in a few weeks. The process of development may then be regarded as *acute* (anterior poliomyelitis, section of a nerve).

As would naturally be supposed, the process is less rapid when a tumour or cicatricial tissue exercises a gradual compression on the nerves, nerve-roots, or anterior horns of the spinal cord. Sometimes the process is *subacute*, and sometimes it is slow, or *chronic*.

In traumatic nerve lesions, the acute development of RD, that is to say, its rapid initiation after the injury, is always suspicious; it always indicates severe injury to the nerve, and the probability is that actual section has occurred. At the same time, acute signs may follow simple trauma of the nerve (violent contusion), severe compression by a fragment of bone, or by a hæmatoma, or purulent infiltration.

The subacute development of RD in gun-shot wounds is generally associated with permanent compression, continued or progressive.

The involvement of a nerve in a mass of scar-tissue, the development of an intrafascicular neuroma, and enclosure in a callus, all give rise to a slow development. For several weeks there is partial RD, which is not, however, always a favourable sign in cases of this kind. As a rule it is merely a stage preparatory to complete RD, into which it will merge insensibly.

Nothing is more deceptive than a case exhibiting partial RD which does not recover within a comparatively short time. Complete paralysis with partial RD may sometimes involve a given region, and after a few weeks the paralysis may completely, or almost completely, disappear, while the signs of RD, on the contrary, persist. After some time, and for no apparent reason, the paralysis gradually reappears in that region. This course of events is usually a sign of inflammatory compression followed by fibrous contraction.

In infective or toxic neuritis, the progress of RD is closely connected with the progress of these affections.

In chronic affections of the spinal cord or its meninges, which effect slow changes in the motor nuclei, or compress the spinal nerve-roots (chronic poliomyelitis, syringomyelia, lateral sclerosis, or any form of chronic meningo-myelitis), its development is more or less slow and gradual.

VII. CLINICAL SIGNIFICANCE OF THE REACTION OF DEGENERATION

Diagnostic Significance.—The reaction of degeneration only appears when a pathological process affects the *peripheral motor neurone or its cellulifugal continuation* (fig. 37); that is to say, the anterior horn cells in the cord (*poliomyelitis*, etc.), their prolongations into the medulla (bulbar motor nuclei : *labio-glosso-laryngeal paralysis*), the motor nerve-roots (*radicular paralyses*), or the peripheral motor neurone (*traumatic changes : section, compression*, etc.; forms of *neuritis*).

Any pathological process not calculated, by its anatomical situation, to affect the peripheral motor neurone, *never* gives rise to the reaction of degeneration. The reaction never accompanies brain lesions, primary affections of the muscles (myopathies, reflex atrophies), or myelopathies localised in the white matter or the posterior horns. Nevertheless RD may appear in the latter if the anatomical change simultaneously affects

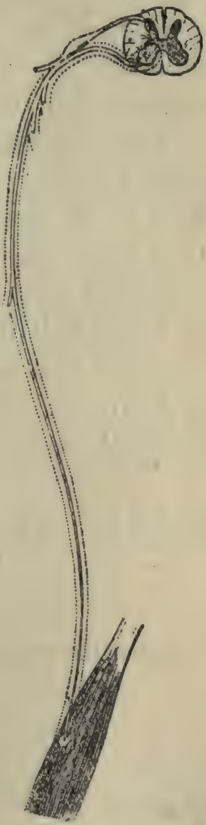

FIG. 37.—Topography of RD. The dotted line encloses the peripheral motor neurone.

the peripheral motor neurone at any point, or attacks it secondarily (as in amyotrophic lateral sclerosis, syringomyelia, and hæmatomyelia).

The existence of the reaction of degeneration in any given case, therefore, introduces *an element of certainty* in the problem of diagnosis, by immediately excluding a number of diseases, and by limiting investigation to the pathological conditions, the site of which correspond to its distribution.

Its absence, however, *does not exclude these possibilities*; because the reaction of degeneration has not been detected, it should not be concluded that the peripheral motor neurone is not involved in the seat of the affection. Poliomyelitis or neuritis, for instance, if their anatomical effects are not far-reaching, may give rise merely to quantitative modifications of electrical excitability, and not, therefore, to RD.

PRINCIPAL AFFECTIONS OF THE NERVOUS SYSTEM IN WHICH RD APPEARS

Brain

Changes in the bulbar nuclei .	Labio-glosso-laryngeal paralysis. Areas of softening. Tumours.

Spinal Cord

Lesions of the anterior horns	*Acute processes*	Infantile paralysis. Acute anterior poliomyelitis in the adult.
	Chronic processes	Forms of chronic poliomyelitis (of the Aran-Duchenne type, etc.).
Lesions invading the anterior horns		Amyotrophic lateral sclerosis. Syringomyelia. Hæmatomyelia.

Compressions at the point of exit of the nerve roots or on their course within the vertebral canal	Pachymeningitis. Tumours. Fractures and dislocations.
Inflammatory lesions of the spinal nerve-roots	Radiculitis in cerebro-spinal meningitis, etc.

Peripheral Nerves (Roots, Trunks, and Branches)

Traumatic changes . .	By division, compression, stretching, etc.
Infective neuritis (*localised or multiple*)	In typhoid, syphilis, gonorrhœa, scarlet fever, influenza, diphtheria, tuberculosis, etc.
Toxic and dyscrasic neuritis (*localised or multiple*)	In poisoning by alcohol, lead, and carbon bisulphide, and in diabetes, etc.
Neuritis *a frigore* . .	Facial paralysis, radial paralysis, etc.

The topographical data afforded by the existence of RD are of the greatest value in establishing or verifying a differential diagnosis.

Its presence during the course of a motor paralysis is absolute proof that this paralysis is not consequent on a lesion of the cortex or of the corona radiata, and is neither hysterical nor simulated. Nor does RD occur in the course of muscular atrophy. The possibility of either reflex or myopathic muscular atrophy may also therefore be eliminated at once.

But although RD enables us to localise the anatomical seat of an affection to the peripheral motor neurone, there is nothing in the syndrome itself to suggest *what part of the neurone* is involved, or in other words, whether the pathological process is located in the cord, the nerve-roots, or the trunk of the pheripheral nerve.

In this connection, however, its muscular distribution may be of some limited value. If, for instance, RD is found in practically the same degree throughout the region innervated by a single nerve, peripheral neuritis may be deduced. If it should be localised to muscles belonging to one radicular group, and cannot be detected in any other muscle, it would be reasonable to suggest a lesion of the anterior horns, or a radicular paralysis.

Significance in Prognosis.—The degree of RD (complete, partial, or traces of RD) is not a " measure " of the gravity of any given case, or a reliable guide in prognosis, *except by comparison with other cases of the same nature.* Thus the muscles of the antero-external region of the leg may be affected in the same degree (by complete RD, for instance) in one case by poliomyelitis, and in another by influenzal neuritis. Notwithstanding the equal degree of RD in the two cases, the prognosis is essentially different, since in the first case recovery is doubtful, but, on the contrary, highly probable in the second.

Similarly, partial RD, which is a favourable sign in facial paralysis *a frigore*, suggests a grave prognosis when it appears in the course of a medullary affection (poliomyelitis or syringomyelia).

With an equal degree of RD, the outlook is often more unfavourable in the case of lesions primarily involving the cells than in lesions of the nerve-roots or nerves. A nerve-root lesion is also generally more serious than a nerve lesion exhibiting an equal degree of RD, on account of its greater tendency to produce secondary lesions in the cells.

The significance of RD in prognosis is therefore always relative, and entirely dependent on the *nature*, *form*, and *etiological conditions* of the affection in which it is observed.

Only for any one given morbid process, and "all other things being equal," to use Erb's appropriate phrase, can the degree of RD serve as a means of comparison, as an indication of the gravity of the lesions, or for the purpose of distinguishing between slight and severe forms.

In poliomyelitis the degree of RD in each muscle reflects the intensity of the corresponding disturbance in the cord, and indicates the chances of functional recovery in each of them. In a peripheral paralysis caused by neuritis of known nature, the degree of RD will indicate the gravity of the condition and its probable duration. There could be no better example in this connection than that of peripheral facial paralysis *a frigore*, in which *there is no clinical sign affording evidence as to the gravity of the condition*, and the degree of RD is the only source of information which we possess.

It is unwise in any other condition to draw definite conclusions as to prognosis from electrical data, (1) *before allowing RD time to develop* (*i.e.* before the end of the second week in acute cases, such, for instance, as traumatic nerve lesions), (2) *before having followed the evolution of RD by repeated examinations*, either in order to decide whether it is proceeding steadily towards the stage of absolute inexcitability, or is tending to remain stationary, or in order to establish the fact that a partial RD, which should be a favourable sign, is not actually developing into complete RD, which would indicate that the causative affection is still an active process.[1]

VIII. The Theory of the Elementary Reactions in the Reaction of Degeneration

It is now generally agreed that the paradoxical combination of *faradic inexcitability* and galvanic excitability in a muscle is connected with the factor of duration of the exciting wave. Currents consisting of short waves, such as the faradic current, lose the power of exciting muscles showing the reaction of degeneration, whereas the galvanic current, with its longer wave, does not. With means of diminishing the duration of the galvanic wave, and with discharges from condensers, the duration of which is a function of their capacity, these facts may readily be verified.

This inability of the muscle to respond to short waves indicates morphological or functional changes which it is the business of electro-physiology to explain.

One of the most attractive theories hitherto forthcoming was formulated by Mlle. Joteyko. It is founded on the double function of the muscle as related to its structure. Muscles with abundant sarcoplasm (smooth muscles) would be only slightly excitable by short waves, and their contraction would be slow. On the other hand, muscles rich in differentiated (fibrillary) substance would respond to the faradic current with a brisk contraction. In pathological conditions giving rise to RD, there would be in some sense a return to the embryonic condition by disappearance of the differentiated substance, and the reactions observed, analogous to those of smooth muscle, would be those of the sarcoplasm.

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[1] The reader will not be surprised at not finding descriptions in this work of certain electrical syndromes, such as the myotonic reaction of Thomsen's disease, the distinguishing reactions in myopathies, and the symptomatic myasthenic reaction of Erb-Goldflam's disease.

These affections are relatively rare in time of peace, and will obviously only occur very exceptionally, if at all, in the neurology of war.

Galvanotonic and sustained contractions would similarly be modes of response of the sarcoplasm.

The production of a slow contraction apart from degeneration, notably by the action of cold, is not incompatible with this theory if we admit that the sarcoplasm retains its excitability at low temperatures, whereas the differentiated substance only reacts within certain limits of temperature.

An objection to this theory, which Mlle. Joteyko fully recognises as valid, lies in the fact that the excitability of the sarcoplasm is assumed, although no experimental proof of its excitability exists. It is difficult, however, to deny the sarcoplasm a property common to all undifferentiated protoplasm.

Another interpretation is founded on histochemical changes in the muscle.

Cluzet succeeded in obtaining experimental reaction of degeneration in a number of ways—by injections of strophanthin and curare, in muscular fatigue, and in experimental anæmia of the cord. Other physiologists have produced a slowing of the contraction in the frog by injecting various substances—caffeine, veratrine, quinine, etc. Similarly, Reiss obtained analogous results by using solutions of potassium and ammonium, and attributes the slowness of contraction observed in diseases associated with cachexia (diabetes and cancer) to a change of toxic origin in the muscle fibre.

A third opinion involves Schiff's conception of the double origin of the muscular contraction. The latter may be produced either by stimulation of the nerve fibres and nerve endings, in which case the response will be a brisk contraction (Schiff's neuro-muscular contraction), or by direct stimulation of the muscle-fibre itself (Schiff's idio-muscular contraction). In the second case the contraction will be slow.

Strümpell, Babinski, and other neurologists have come to regard the question in this light, and regard the phenomena of the reaction of degeneration, and particularly the slow contraction, as the normal response of a muscle separated from its motor nerve. "The reaction of degeneration," writes Babinski, "is, at least in part, nothing more than the reaction proper to the muscle fibre." [1]

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[1] Babinski, *Trav. scient.*, Paris, 1912.

This theory, however, complicates the explanation of partial reaction of degeneration, since notwithstanding the preservation of neuro-muscular excitability to the faradic current, the muscle responds by a slow contraction to the galvanic.

Lapicque [1] returns to the conception of a loss of differentiation in the muscle, following a nerve lesion, and consequent anatomical and functional similarity to smooth muscle. This physiologist discards the sarcoplasm hypothesis, and confines himself to a consideration of the slowing of the contraction, which necessarily corresponds to a diminution of the quickness of excitability. Faradic inexcitability is explained by the fact that the faradic wave is now too short for the slowed excitability, whereas the galvanic current exercises its effect throughout the duration of the excitable phase, just as in the normal state.

The inexcitability of the nerve is due to the fact that its rapidity of excitability remains unaltered, whereas that of the muscle is reduced. Now in the normal state the rapidity of excitability in nerve and muscle are equal: their *chronaxie* (see p. 111) is the same. This isochronism is the condition of transmission of the stimulus from the nerve to the muscle, and this transmission is impossible, if, as in a pathological condition, the rapidity of excitability of the muscle is reduced.

Polar inversion has also been differently explained. Wiener observes that, in a muscle undergoing degeneration, excitability gradually diminishes at the motor point, and its maximum is thus apparently shifted towards the extremities of the muscle. Under these conditions a stimulus applied at the motor point would be ineffective, and the muscle would respond to the virtual pole of opposite sign which excites it at its periphery. In support of this theory, Wiener showed that inversion can readily be produced in the gastrocnemius of the frog by alterations in the muscular tissue brought about by certain solutions. The physiological law of contractions is therefore perfectly compatible with RD, and inversion is merely an apparent modification connected with topographical changes in excitability. Wiener's hypothesis has the advantage of explaining at the same time both the displacement of the motor point and the habitual preservation of the normal formula in longitudinal excitation.

It is difficult, however, to understand why certain cases of partial RD, in which preservation of the excitability of the motor nerve indicates a relatively moderate degree of degeneration, should be accompanied by inversion, and conversely why cases of complete

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[1] Lapicque, *Communication orale.*

RD, exhibiting advanced muscular degeneration, should present the normal formula. These are contradictory facts which the theory leaves unexplained.

The sarcoplasm theory is an explanation of polar inversion. Smooth muscle would normally exhibit inversion of the formula. Pflüger's law would be reversed in the case of muscles rich in sarcoplasm. The closing stimulus for undifferentiated protoplasm would be produced at the anode (Mlle. Joteyko). Degenerated muscle, coming to resemble smooth muscle in structure, would consequently obey the same law.

This theory, however, is also open to a definite objection. How, in fact, can it be explained that polar inversion, if it is to be considered characteristic of a muscle undergoing degeneration, is not more constant, and in longitudinal excitation rarely if ever present ?

It will be seen that the question is extremely complex, and that all the suggested explanations have their weak points.

Is the question, after all, one of chemical change in the muscle ?

In animals, particularly in the frog, the normal formula may be varied by impregnating the muscles with solutions of potassium or ammonium. On the other hand, solutions of sodium and lithium cause reversion to the normal formula. It has therefore been debated whether a modification in composition of the cell protoplasm, such as occurs in the reaction of degeneration, would not have the effect of rendering the action of the positive pole more powerful than that of the negative. According to Reiss, the results obtained experimentally by Wiener can best be explained in this way, since in impregnating the muscle with substances affecting the excitability of the motor point, he cannot have prevented their diffusion, and assuming the absorption of toxic substances by the muscle fibre, his theory is virtually equivalent to the theory of chemical change.

IX. THE ADOPTION OF A UNIFORM TECHNIQUE

Following this study of abnormal electrical reactions, we propose discussing the question of the ambiguity of terms used in electro-diagnosis. What, for instance, is understood by *great faradic, or galvanic, hypo-excitability*, expressions which are currently employed in reports for medical boards ? Beyond what limit in the strength of current used does galvanic hypo-excitability become "great"? When faradic inexcitability is deduced, was the coil employed one of fine or coarse winding, and were the results obtained by the unipolar or bipolar method, with single shocks or the tetanising current? And was there actually inexcitability to all these forms of stimulus?

It certainly seems that, were an understanding arrived at between electrologists, a greater degree of precision would be a simple matter; the terminology of electro-diagnosis would become a uniform language, capable of expressing reactions and their development in more exact terms, and obviating futile scientific discussions as to the value of different reactions described under the same name.

With a view to such an understanding, we suggest the following standards:

(*a*) *Testing with the Faradic Current.*—It should be understood that all faradic tests are carried out with a standard type of coil and a current of 4 *volts* in the primary circuit, with a secondary coil *of fine wire*, employing the *tetanising* current, by the *bipolar* method. We need hardly emphasise the importance of a uniform voltage in the primary circuit for comparing degrees of excitability. A coil wound with fine wire is employed because strong currents are sometimes required in testing nerves and muscles in a state of hypo-excitability.

With standard apparatus, the measurement of variations in excitability in terms of the distance between the two coils, although illogical, would be a satisfactory system.

As has already been mentioned, it sometimes happens that a nerve or muscle, which has become inexcitable by the tetanising current, may yet react more or less to single shocks. It would therefore be well to agree not to deduce faradic inexcitability unless the nerve or muscle has been tested by both of these methods, with a negative result in each case. The expression *integral inexcitability* might well be adopted in future to indicate that under the technical conditions described above, excitability is abolished both for tetanising current and single shocks.

(*b*) *Testing with the Galvanic Current.*—1. It should be understood that when inversion is described and no details are specified, inversion *at the threshold* is referred to: strong currents should be mentioned, if inversion is produced by them and not otherwise.

2. Although the character of the contraction is a matter of personal observation, we consider that three degrees should be distinguished: *just perceptible slowing*, *slow contraction*, and *very slow contraction.* The last expression should refer to cases in which the slowing resembles the sluggish reactions observed in lead paralysis.

3. Describing a slow contraction as *unipolar* should be understood to mean that the slowing is only observed at anodal closing, a common circumstance in early stages or mild forms of RD, as has already

been mentioned. The absence of this term should signify that the contraction is slow at both poles.

4. The meaning of the terms *relative longitudinal hyper-excitability* and *absolute longitudinal hyper-excitability* should be defined as already stated (see p. 65). To describe the combination of *abolition of the excitability at the motor point* and *preservation of longitudinal excitability*, we suggest reviving a term which has fallen partially into disuse, *viz.* the *remote reaction of degeneration*, or DRd.

5. By the term *inexcitability* it should be understood that the muscle is no longer excitable by currents which can be tolerated, either at the motor point or by longitudinal excitation.

If excitability be recovered when the indifferent electrode is brought near to the active electrode, the words "*except with the bipolar method*" might be added.

"*Inexcitability, including bipolar*," would infer the absence of response to any current that could be tolerated.

X. ELECTRO-DIAGNOSIS WITH CONDENSERS

In the course of several preceding chapters we referred to the imperfections of the faradic apparatus, particularly the use of varying and un-standardised instruments, instanced by the archaic but still existing method of measuring intensity in units of distance.

Galvanic technique is also open to a good deal of criticism. Thus, when we read on the milliamperemeter what we call the liminal intensity, which we have adopted as a measure of excitability, we are merely recording an apparent intensity: in the first place, the skin masks very slight muscular contractions, and we are therefore not measuring the actual threshold; further, since the current diffuses from its point of entry into the body, the zone of the motor point is only affected by a fraction of the total intensity; the action of the neighbouring tissues in regard to this fraction, in itself effective, is that of a shunt. Further, the intensity recorded by the milliamperemeter depends on various factors—the resistance of the circuit, and polarisation in the tissues. Lastly, as was observed by Doumer, when the circuit of stimulation is made, the fall in resistance produced by the passage of the current, only the maximum of which is indicated by the milliamperemeter, prevents our observing the exact moment, and consequently the actual intensity, of the effective stimulus.

Without going as far as to urge, with Doumer, that time spent in this way is absolutely wasted, we cannot but agree that the present method, the farado-galvanic, is far from being exact, and that by it we can only expect to obtain rough measurements, which in practice are only of value when marked changes have already taken place.

To ensure greater accuracy, a number of electrologists have attempted substituting the use of condensers for the classical method.

The condenser, which had already been used extensively in physiology (Chauveau), was first employed in a systematic way for purposes of electro-diagnosis by Czybulski and Zanietowski.

1. **Condenser Method with Variable Charge.**—We have seen that the duration of the discharge from a condenser is proportional to its capacity. Consequently, if two condensers of different capacity be employed, one giving a short wave of discharge and the other a long wave, the system is analogous to the farado-galvanic, the short wave corresponding to the faradic wave and the long wave to the continuous current wave.

In practice, in the method "*of charging to variable potential*," four condensers, of 0·01, 0·1, 1, and 10 microfarads are used, and for each capacity the voltage is increased until the liminal voltage is obtained. Thus, for instance, tests for excitability are first carried out with 0·01 microfarad. If the maximum available voltage (80 to 110 volts) gives a contraction with this low capacity, the capacity of 10 microfarads can at once be used for obtaining stimuli with long waves. In cases in which the muscle does not respond to 0·01 microfarad, 0·1 microfarad is tried. When the liminal voltage has been recorded, further tests are carried out as before, with 10 microfarads. If 0·1 microfarad, corresponding to short waves, is without effect with the maximum voltage, 1 microfarad is used, and so on.

Apart from the fact that the testing apparatus is constant, the principal advantage of condensers is that they permit a more accurate estimation of variations in excitability with short waves. The delicacy of the method enables the neurologist to follow variations in voltage, and consequently changes in the electrical reactions, more closely than the farado-galvanic method permits.

In spite, however, of its undeniable advantages, this method is open to objection, in that it is somewhat laborious. It is generally regarded as too complex, and comparatively few have become adepts with it.

2. **Method of the Condenser of Variable Capacity.**—The factor of the *duration* of the stimulus, the import-

ance of which will be seen in the next paragraph, suggested to Cluzet an entirely different method from that of fixed capacities, founded on the employment of *a condenser the capacity of which could be regulated.*

Described by the author for the first time in 1911,[1] this method is in use at the electro-radiological centre at Lyon, and in several secondary centres in the 14th Region.

Its principle consists in determining the lowest capacity, that is to say the shortest wave, giving the threshold of muscular contraction.

As in ordinary electro-diagnosis, the method concerns itself with the form of the contraction, the relative action of the two poles, and the longitudinal reaction.

As it is possible to utilise a long series of waves intermediate between the short wave given by the lowest capacity (equivalent to the faradic wave) and the long discharge (analogous to the galvanic current), a whole series of degrees of RD can be distinguished in muscles exhibiting the reaction, which could scarcely be differentiated by the farado-galvanic method.

In certain cases of very marked RD, the largest condenser, the duration of the discharge of which is too short, produces no effect. Under such conditions, instead of employing a greater capacity, which would render the apparatus bulky and cumbersome, an additional resistance is introduced into the circuit, the effect of which is to lengthen the duration of the discharges without affecting the actual quantity of electricity.

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[1] Cluzet, "Méthode et appareils nouveaux pour l'électro-diagnostic," *Lyon Méd.*, Nov. 26th, 1914.

The following is a brief summary, adapted from Cluzet, of the technique of the method employing the condensers of variable capacity.

In the first place, the value of the normal liminal capacity with the voltage used, is determined by means of the apparatus described on page 14. With 110 volts, normal nerves and muscles usually respond to approximately 0·02 microfarad, the indifferent electrode being 100 sq. cm. in area, and the active electrode 1 sq. cm.

Hyper-excitability and hypo-excitability are then indicated by the relation between the capacity giving the threshold and the capacity of 0·02 microfarad.

In the case of hypo-excitability the slowness of the contraction will indicate the reaction of degeneration.[1] If the muscles exhibiting this hypo-excitability, together with slow contraction, are still excitable indirectly through the motor nerve, partial RD will be deduced. If they are not, it is a case of complete RD.

When no response can be obtained with any of the capacities, additional resistances are successively introduced into the circuit. Only after this final test can the absence of contraction be held to indicate inexcitability of the muscle.

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[1] It should be observed that the condenser never gives the galvanotonic reaction.

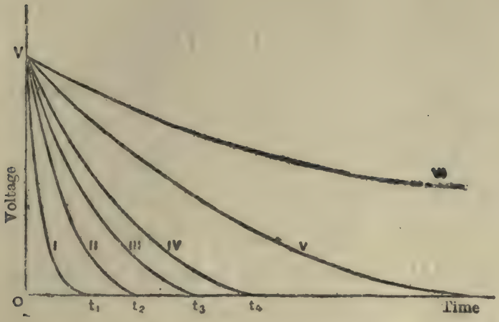

FIG. 38.—I, II, III, IV, etc., are curves representing the discharges of condensers of increasing capacity charged to the same potential (V). The time intervals Ot_1, Ot_2, Ot_3, are proportional to the capacities of the condensers which have given the curves I II, III, etc.

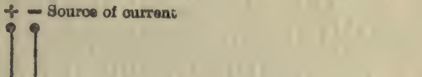

FIG. 39.—Condenser method. The condenser is connected to the source of supply when the Morse key is depressed. It is discharged through the patient when the key resumes its position of rest. At E, the active electrode; at E′, the indifferent electrode.

XI. TRUE EXCITABILITY—ITS MEASURE, THE CHRONAXIE

The Law of Excitation and the Chronaxie.—The criticisms brought against the current method in electro-diagnosis to which we have referred above are of small importance by comparison with a fundamental objection of a different nature. The electrical diagnosis of the present day is based on the assumption *that the property of tissues known as excitability may be measured in terms of intensity.*

Now although it is true that in practice the measurement of excitability in terms of liminal intensities, a method which has been general since the days of Duchenne and Erb, is daily of the greatest clinical value, is not this system of measurement purely conventional ? Is excitability really and exclusively a function of the intensity ?

Is it not also peculiar that measurements of two forms of excitability, faradic and galvanic, should have to be carried out, where only a single means of stimulation is concerned ?

As far as the theory of excitability is concerned, the problem has to a great extent been solved by the work of certain present-day physiologists.[1] The difficulties of measurement in practice, however, have not yet been overcome.

The absence of response to the faradic current, *i.e.* to short waves, in certain pathological conditions of the nerves, might have been expected to call more attention to the importance of the factor *duration* in the phenomena of stimulation.

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[1] See in particular : Lapicque, *Soc. de biologie*, 1909, and *Revue générale des Sciences*, Feb. 1911, and Laugier, *Thèse de Paris*, 1912.

Engelmann[1] was the first of the physiologists to comprehend the influence of the time occupied by the passage of the current on the phenomena of stimulation, and to regard the *physiological time* as a property of the tissue.

It was not until much more recently (1891) that Hoorweg stated the law establishing the relation between the two factors, *duration* and *intensity*. He observed further, with the condensers which he was using, that with a certain kind of discharge, that is to say with a stimulus of a certain duration, the energy reached a minimum. The corresponding duration was subsequently considered by Waller as a characteristic of the tissue stimulated.

The study of the law of excitation and the effect of the duration of the passage of the current was again undertaken by Weiss. Instead of employing waves of discharge from condensers (Hoorweg), Weiss worked with rectangular waves obtained by the very rapid make and break of a continuous current. He deduced the following law from the results of his experiments.

An electrical stimulus is effective only when a constant quantity of electricity is employed, increased by a variable quantity proportional to the duration of the discharge.

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[1] Engelmann, "Beitr. z. allg. Muskel. u. Nervenphysiol.," *Pflüger's Arch.*, t. III and IV.

This law is expressed by the following formula:

$$Q = a + bt$$

where Q represents the quantity of electricity employed, t the duration of the current, and a and b two coefficients peculiar to the tissue concerned.

The exact significance of these two coefficients was at first not understood. Zanietowski regarded the coefficient a as representing the initial quantity of electricity required to charge the capacity represented by the tissues, and the factor b as the intensity (varying with the resistance of the tissues and electrodes) of the continuous current passing through the body. It at once became evident that a and b underwent the same variations in all the experiments carried out, and consequently that their relation remained practically constant.

Since, in Weiss's formula, the coefficient a is a *quantity*, and the coefficient b an *intensity* (since bt, product of an intensity and a time, is a quantity), the quotient $\frac{a}{b}$, the quotient of a quantity by an intensity, must of necessity be a time. This time $\tau = \frac{a}{b}$ is therefore, in the same way as a and b, a characteristic of the tissue: it is a *time constant*, independent of experimental conditions.

It is high in the case of muscles of which the contraction is slow, low in those which contract rapidly, varying inversely with their *rapidity of excitability*, and corresponding exactly to Engelmann's "physiological time." This is the *chronaxie* of Lapicque.

Briefly, whereas the measurement of excitability by the ordinary determinations of intensity is purely arbitrary and without physiological basis, that of the relation $\frac{a}{b}$ gives a constant, proper to the organ concerned and independent of any other factor.

Measurement of the Chronaxie.—The measurement of the chronaxie has been carried out in physiology by several means. The simplest procedure (Lapicque) consists in determining the duration of the stimulus required with an intensity twice that which will give the threshold contraction when a current of prolonged duration is used.[1] Hitherto, however, it has not been possible to apply any of the procedures employed in physiology, to man.

The importance, however, from the clinical point of view, of possessing a simple method by which the rapidity of excitability of affected nerves and muscles could readily and rapidly be determined, will at once be understood.

What has been called the "*effective time*" can be estimated to some extent by Cluzet's method of condensers of variable capacity, already described. The results obtained vary as the chronaxie, and the method may therefore be regarded, if not as a definite measurement, at least as affording an indication.

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[1] This intensity of a current of indefinite duration giving the threshold was designated by Lapicque the "rheobase."

In the same way the procedure of obtaining the *indices of rapidity* (Bourguignon and Laugier) cannot be said to give a measure of the chronaxie, but affords corresponding indications.

The principle of the Bourguignon-Laugier method is to determine the relation between the quantities of electricity required for a liminal make and a liminal break stimulus, in the case of a given nerve or muscle, the same apparatus being used in each case. It has been shown, by the work of Mmes. Lapicque and J. Weill, that the relation between the quantities corresponding to the two waves, classes nerves and muscles in the same order as their rapidity of excitability.

In applying the method, a coil is used which will readily give both thresholds ; and the intensity (in cms.) of each of the two waves which gives the threshold is determined by successive increases in the closing current.

The opening contraction is eliminated by a special interrupter while the closing contraction is being tested for, the opening stimulus being extremely painful to the patient in cases where the threshold for closing stimuli is considerably raised.

If the equivalents, in quantities of electricity, of values in centimetres of approximation, have already been determined (by means of a ballistic galvanometer), reference to these will give the quantitative relation desired.

This relation is constant for any given muscle ;

for the majority of normal muscles it is in the neighbourhood of 15. In pathological conditions it falls more or less below this figure.

The only disadvantage of the method is that it can only be applied to cases in which faradic excitability has not been lost. In other words, it cannot be used in complete RD. It is true, on the other hand, that it can be used to follow the early evolution of RD very closely, and that it is applicable to all cases of partial RD.

The *direct measurement* of the chronaxie was attempted by Cluzet with the condenser, and effected by Lapicque by means of his chronaximeter.

Cluzet's procedure consists in determining which of several capacities giving the threshold will give it with a minimum of energy.

A potential-reducer for varying the charge and a voltmeter to record the potential of the charge are placed in the circuit.

The threshold voltage is determined for a number of capacities. The energy expended in each of these cases is calculated ($E = CV^2$), and the lowest figure gives the *optimum capacity* C.

The measure of the characteristic of excitability is then obtained by the formula :

$$\text{Characteristic} = RC$$

which, as will be seen, necessitates measurement of the resistance.

Lapicque's *clinical chronaximeter* is a rotatory rheotome recording directly the passage of currents in terms of absolute time, which may be regulated at will from a thousandth to a tenth of a second.

The apparatus consists of a motor system and an interrupter system.

The motor system is actuated by the fall of a weight P. The cord by which this is suspended is wound on a spiral conical pulley, the axle of which therefore revolves with an acceleration which equals the square of the time. A needle A, mounted perpendicularly on this axle, describes an arc which records the graduation of the apparatus in fractions of a second.

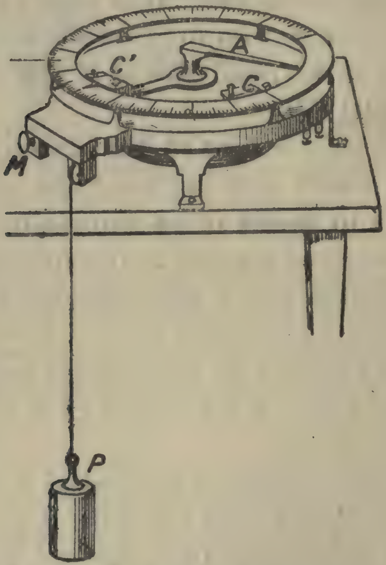

FIG. 40.—Lapicque's chronaximeter.

By engaging the levers C and C^1, this needle successively makes and breaks the ordinary galvanic circuit of an electro-diagnosis table, in which the apparatus is placed. By increasing or decreasing the distance between the two mechanisms of make and break, the lapse of time between the two waves, or, in other words, the duration of the current, can be varied at will.

To use the chronaximeter, the galvanic threshold is first tested for with Courtade's key; the apparatus is wound up, and then released. Should no contraction occur, the duration is too short. By gradual further separation of the contacts, a point is reached at which a contraction is produced. The contacts are then approximated until no further contraction occurs.

This minimum distance between the contacts is then read off on the circumferential scale of the apparatus.

This figure, however, does not correspond to the chronaxie itself, since the apparatus measures, not the chronaxie, but the "effective time."

The effective time is the minimum time for which the key of an interrupter must be kept down, in order to obtain the threshold. It is a multiple of the chronaxie, roughly equal to ten times the value of the latter.

The effective time for the human muscle is about 0·002 to 0·003 seconds (chronaxie: 0^{σ},2 to 0^{σ},3), which corresponds to close approximation of the two contacts.

In the pathological state the distance between them is increased.

After a nerve lesion, a muscle exhibiting signs of definite degeneration by ordinary methods gives an effective time of one-tenth of a second (chronaxie : 10σ) or even longer, *i.e.* fifty times greater than the normal.

In cases of partial RD, intermediate figures are given, indicating chronaxie of 1 or 2σ to 10σ, according to the degree of degeneration.

The chronaximeter fills an important place in electro-diagnosis. The simplicity of its construction and the ease with which it can be manipulated are a guarantee for its future. Introduced in 1915 by Lapicque, its use is being studied in many laboratories.

CHAPTER III

ELECTRO-DIAGNOSIS IN MOTOR PARALYSES

I. TRAUMATIC PARALYSES OF NERVES

THE motor paralyses due to changes in the peripheral nerves which occur in the pathology of war are due, either to traumatic or to toxic and infective causes.

The latter, with which may be included the paralyses of hitherto undiscovered cause referred to as rheumatic, or *a frigore*, are of a purely medical character, demanding medical and physiotherapeutic treatment, and do not involve the complex problems of traumatic paralyses. The electrical reactions to which they give rise will be studied in a later chapter.

In the category of traumatic paralyses we shall introduce in the first place paralyses from section, contusion, and stretching;[1] and secondly, paralyses from continuous and prolonged compression, whether of exogenous origin (aneurysm, hematoma, abscesses, splinters of bone, callus, involvement in scar tissue, fibrous bands, etc.), or of endogenous origin, as in traumatic neuroma.

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[1] Further causes in this category are burns, irritating injections of ether, alcohol, cocaine, and camphorated oil, either into the nerve or in its immediate neighbourhood.

This etiological classification, though necessarily incomplete, is clear and convenient, but does not always correspond with clinical experience.

Gunshot wounds are often complicated, and the cause of the nerve changes is not certain. In open wounds, for instance, whether suppurating or clean, and in compression by purulent accumulations, we cannot determine the extent of the part played by infective neuritis, which is sometimes latent, in the process causing paralysis. Similarly, surgical intervention has shown that partial division, neuroma, and involvement in a mass of scar tissue, are often present together in the case of the same nerve.

It will therefore be understood that each of these conditions may exercise varying degrees of effect on the electrical reactions.

Differential Diagnosis.—Differentiation between the traumatic nerve lesions mentioned above, division, contusion, and compressions of all kinds, and pathological conditions of a hysterical, reflex, or imaginary nature, offers no difficulties when marked changes in the electrical reactions have occurred. The discovery of profound alterations in electrical excitability, and, *a fortiori*, of the existence of RD, affords definite information, the value of which calls for no further discussion.

There is only room for doubt when the electrical reactions are only slightly modified: it should be remembered that *the absence of abnormal reactions is not absolute proof that the nerve is unaffected.* Huet[1] has described slight irritative forms, the organic nature of which is established by the distribution of the muscular atrophy, the presence of definite painful points, and sometimes even by an absent or feeble tendon reflex in a case where electrical examination reveals no appreciable alteration in excitability of either nerves or muscles.

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[1] Huet, "Les troubles de l'excitabilité électrique des nerfs et des muscles" *in* M. et Mme. Déjerine et Mouzon, *Presse médicale*, August 30th, 1915.

Electrical testing may seem superfluous in many cases, since clinical methods, embracing a number of cardinal signs and a large variety of slighter indications which recent work has afforded the neurologist, generally suffice to differentiate between organic and functional paralyses.

In spite of this, the electrical method should invariably be applied, in the first place because it verifies previous conclusions, an additional safeguard against ever-present possibilities of error; further, because it may undeniably lead, in some cases, to reconsideration of a hasty or even apparently well-founded diagnosis; and finally because, where organic lesions and functional disturbance coexist, it is almost the only means we have of defining the extent of each of these.

Similarly, when several nerves are affected at the same time, the electrical reactions indicate in what degree each is involved.

Evidence afforded by Electro-Diagnosis as to the Nature of the Lesion.—In the case of a wound, when the possibility of a non-organic cause has been eliminated, the principal question is that of the nature of the lesion (division, compression, or irritation), since it is upon this that the form of treatment depends (physiotherapeutic, surgical, or expectant).

The most serious possibility in a motor paralysis is complete division of the nerve, a condition which calls for surgical intervention and suture.

Now numerous observations extending over the last few years have not educed any infallible clinical sign of nerve division. Similarly, the syndromes of interruption and compression, solidly founded as they are, admit of such considerable error that conclusions drawn from them can only be provisional.

The numerous smaller signs recently described, such as vaso-motor disorders, absence of sweat reactions, hypotrichosis, and cicatricial topo-paræsthesia, are considered valuable indications of a serious nerve lesion, but afford no evidence as to its nature.

The evidence obtained by electrical methods is similarly indefinite, but taken in conjunction with the clinical signs it is of such importance that no observation omitting reference to the electrical reactions can be regarded as complete.

Evidence of division of the nerve in man is afforded by the complete reaction of degeneration. Its characteristics require no further description, as this particular lesion was taken as the type in the study of complete RD and its evolution.

Every case of nerve section is therefore characterised by complete RD. The converse, however, is not true; the existence of RD does not necessarily imply that the anatomical continuity of the nerve is broken, or that the lesion is incapable of repair. Compression and neuritis may also give rise to complete RD.

Under these conditions, what is there in the intrinsic characters of the reaction of degeneration to indicate the nature of the causative lesion ?

Significance of the Rapid Evolution of RD.—A fact of which sufficient account is not taken in electro-diagnosis is expressed in the following proposition: RD is always in continuous evolution, and the syndrome of that name is merely a *transitory manifestation*.

The elementary reactions which, taken together, are said to constitute the reaction of degeneration, characterise the complete reaction, that is to say RD in its typical phase.

Now this typical phase is preceded by an initial period, and followed by a terminal period. Obviously the evolution of the causative lesion will control the evolution of RD, and much can consequently be gained from an analysis of the latter.

A single electrical examination, revealing partial or complete RD, can never give an adequate idea of the process at work. If the full possibilities of electro-diagnosis are to be utilised, and a conclusion as to the nature of the paralysis is to be founded on as complete a series of indications as possible, it is essential to follow the early stages in the evolution of RD as closely and exactly as possible.

RD is, in fact, best compared to the temperature in pyrexia. The nature of a fever is not suggested by simply taking the temperature once; obviously, to any intelligence, its diagnostic value lies in its variations, and therefore in the whole chart.

The practical corollary of this comparison is that it is absolutely necessary, in cases of peripheral nerve lesions, to undertake repeated electrical examinations; in fact, to carry out a series of examinations at regular intervals. The most interesting and important phenomena are possibly those developing early, during the first two weeks following the injury. At this period an initial examination is particularly instructive. It is therefore important that any patient suffering from a motor paralysis should immediately be sent where a weekly examination by an experienced electrologist could be combined with the treatment of his wound. The presence of a wound is no obstacle to such an examination, since it could be carried out under perfectly aseptic conditions.

To determine whether different electrical phenomena follow division and compression of a nerve, it was natural to resort to experimental methods. We accordingly resected 2 cms. of the right sciatic in a dog, and compressed the left sciatic with a non-absorbable ligature. In spite of the serious nature of these lesions, the animal remained paralysed only for a few days. At the end of the first week it appeared entirely to have recovered the use of the two paws.[1]

The electrical reactions were tested every week for about five weeks; on two occasions the nerve and muscle were exposed and stimuli applied directly. The following were the principal observations:

On the right side (nerve divided), the peripheral end of the sciatic was no longer excitable. Stimulated at the motor point, the internal portion of the gastrocnemius (which was selected for testing) showed towards the tenth day a marked galvanotonic contraction, without inversion of the formula. Stimulated in the region of the tendon, hyper-excitability appeared rapidly, with slow contraction and inversion. Later, excitability diminished at the motor point. The galvanotonic contraction persisted, but was only given by stronger currents. Inversion of the formula occurred. The manifestations of longitudinal excitability persisted in the form of a slow contraction with inversion.

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[1] This paradoxical phenomenon is well known to physiologists, and indicates a peculiar power of rapid replacement and adaptation in the dog.

On the left side (compression), the nerve could still be excited above the lesion. The contraction was at first only tonic at anodal closing; later, both poles gave a galvanotonic contraction. Hyper-excitability was shown particularly by the great ease with which the KC contraction was obtained. Inversion was not observed at any period. In longitudinal excitation, the KC contraction appeared slow and the AC tonic: there was no inversion, but a nearer approach to equality of the KC and AC thresholds than normally.

Briefly, compression was followed by a slower evolution, a more marked stage of hyper-excitability, and the absence of inversion.

It goes without saying that the reactions observed in this experiment would not necessarily be reproduced in similar experiments. They would chiefly depend on the nature and degree of the compression, developing differently in cases of slight compression, whereas in rapid and severe compressions they would be practically equivalent to experimental section, and give rise to an electrical syndrome identical with that produced by division of the nerve.

In the case of wounds, immediate section, that is to say by a projectile in action, is followed by a *rapid evolution* of the electrical reactions.

The initial phase of RD (characterised by progressive diminution of faradic and galvanic excitability in the nerve, progressive diminution of faradic excitability, galvanic hyper-excitability, and then hypo-excitability, in the muscle, and a tendency to slower contraction), assumes an acute aspect, and extends over two or three weeks. At the end of this time the typical phase of the complete reaction of degeneration appears; the nerve has become inexcitable, and the muscle does not respond to the faradic current, but the galvanic causes a contraction which is either slow or tonic.

This acute evolution, taken in connection with the theory of its causation, observation of the track of the projectile, the manner of onset of the paralysis (complete immediate paralysis), and the initial pain (fulgurant pain), suggests a strong probability that the nerve has been severed.

It is nothing more, however, than a *probability*, for the same acute syndrome may follow simple violent contusion of the nerve, or severe compression by a splinter of bone or hæmatoma.

More commonly the case is not examined until several weeks or months afterwards, and if, at this time, the patient no longer remembers the early circumstances connected with his wound, the problem is a much more difficult one. There is nothing to indicate whether the paralysis follows from division of the nerve, severe and sudden compression, slow compression, or neuritis. It is impossible to reconstitute the evolution of the electrical reactions. We are faced with RD in its typical phase, and none of its characters indicates its mode of development, whether acute or slowly progressive.

In animals, after section of a nerve, the progressive evolution of RD terminates ultimately, after several months, in absolute inexcitability. The muscle ceases to respond to longitudinal stimulation and so on, even with the strongest currents.

In man, absolute inexcitability, observed some weeks after the injury, is an extremely serious sign, indicating severe lesions of the nerve and muscle, but not necessarily implying severance of the nerve.

On the other hand, persistence of excitability in the muscle (longitudinal excitation) for several months, does not always mean that the nerve has not been severed. In man, the typical phase of RD is continued for much longer than in the animal, and the stage of inexcitability may not be reached for many months.

The Reaction of Conductivity.—The physiologist Schiff, in experimenting on nerve compression, observed that nerves which were no longer excitable by electric stimuli applied below the point of compression, continued nevertheless to respond to stimuli above that point.

This physiological phenomenon of the individual character of the two nerve functions, excitability and conductivity, is seen again, as we have noted, under different conditions—those of electro-diagnosis. Erb in particular, emphasises it strongly in connection with regeneration of the nerve (see p. 139).

The peculiar condition of continued excitability above the lesion in nerve injuries was recognised by Claude, Vigouroux, and Dumas,[1] who observed it in a number of their cases. Battez and Desplats,[1] however, availing themselves of Schiff's discoveries, have been chiefly instrumental in proving the diagnostic value of conductivity, basing their conclusions on numerous observations.

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[1] Claude, Vigouroux, and Dumas, "Cent cas de lésions traumatiques des nerfs des membres," *Presse Méd.*, March 4th, 1915.

If two faradic electrodes be applied close together immediately above the lesion, in a nerve whose excitability below this point is abolished, stimulation may be ineffective, or, on the other hand, it may provoke contraction in muscles situated distal to the lesion. In the latter case, the nerve, although its excitability is lost, has not lost its conductivity. This persistence of conductivity is *absolute proof that the continuity of the nerve is not broken*, and renders the diagnosis of section inadmissible. Here again, however, the converse is not true ; absence of the conductivity reaction does not necessarily mean that the nerve is severed.

Battez and Desplats observe that the reaction of conductivity may be tested for by bipolar faradic stimulation, and when this is without effect, by *bipolar galvanic stimulation.* This procedure revealed the reaction of conductivity in several cases which gave negative results on faradic stimulation, and in which, therefore, the continuity of the nerve would not otherwise have been proved.

The Reaction of Inversion.—It follows theoretically from the results of experiments that inversion is an integral part of the syndrome following complete nerve section. It appears also to have been observed in all actual cases of complete section, as verified by operation.

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[1] Battez and Desplats, " Excitabilité et conductibilité dans les traumatismes du nerf par blessure de guerre," *Bull. Ac. Méd.*, January 18th, 1916.

According to Bourguignon, when there is no inversion *at the threshold*, the hypothesis of severance should be abandoned.[1]

Inversion is not, of course, a specific reaction in nerve section; it is observed in other cases (exogenous and endogenous compressions).

The Distribution of the Reactions.—Another sign of complete severance is that the electrical reactions are modified in the same degree throughout the region supplied by the nerve. In cases of partial severance, however, and in certain compressions, the abnormal reactions are distributed unequally among the different muscles; in the latter case, the faradic reactions of the muscles are usually absent; but whereas certain muscles give a brisk contraction on galvanic stimulation, in others the contraction is slow. Pierre Marie, Gosset, and Meige[2] have shown that the fibres supplying different groups of muscles may be isolated by direct excitation. It will be evident that partial severance, or a neuroma in contact with the nerve, may affect certain muscles to the exclusion of others.

This sign is of particular value in injuries of the median and ulnar nerves, which are frequently connected with multiple wounds; its value is less in lesions of the radial and external popliteal (Mme. Benisty [1]).

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[1] "Les procédés modernes d'électrodiagnostic," *Cong. de l'A. F. A. S.*, Le Havre, 1914.

[2] P. Marie, Gosset, and Meige, "Les localisations motrices dans les nerfs périphériques," *Bull. Ac. de Méd.*, December 28th, 1915.

Differential Signs in Severe Lesions.—It will be understood from the foregoing that there is no definite and specific sign indicating the nature of the nerve lesion. Conclusions can only be drawn from a group of reactions, certain of which are definite evidence that the nerve is not severed, and others strongly suggest that it is.

A. *Reactions indicating continuity of the nerve:*

1. Incomplete RD or modifications of excitability (quantitative only).
2. Absence of one of the secondary signs of complete RD; absence either of longitudinal hyper-excitability or of slowing of the KC contraction at the motor point.
3. The reaction of conductivity.

B. *Reactions suggesting* (but in no case proving) *division of the nerve.*

1. Acute evolution of RD.
2. Absence of the reaction of conductivity.
3. Inversion at the threshold.
4. Distribution of the same degree of RD throughout any given region.
5. Persistence of hyper-excitability only in longitudinal excitation.
6. Rapid development of absolute inexcitability.

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[1] Mme. Athanasio Benisty, *Traitement et restauration des lésions des nerfs*, Paris, Masson, 1917.

In reality, apart from cases in which the development of the reactions is acute and immediate, accompanied by a clinical history suggesting that severance has occurred, the reactions enumerated in the second category indicate the existence of a severe lesion of the nerve, functionally equivalent to actual severance.

Neuromas and severe compressions may give these reactions in the same way as division of the nerve, and consequently the question of the nature of the lesion may remain undecided; such a case, where electrical tests reveal a serious state of affairs, requires operative treatment, at least of an exploratory nature.

On the other hand, the reactions in the first category, indicating continuity of the nerve, and more particularly suggesting exogenous or endogenous compression or neuritis, afford a reasonable hope of spontaneous recovery, and indicate appropriate medical treatment. Only in the case of aggravation of the clinical and electrical signs is an operation necessary.

Discrepancies.—In several cases it has been observed with some surprise that the electrical reactions do not appear to correspond with the clinical signs or with the gravity of the lesion revealed by operation.

In two cases, for instance, of sciatic paralysis with marked RD, Basset,[1] after minute examination, could find no visible lesion of the nerve. In regard to the possible objection that this lesion might have escaped him, he avers that the macroscopic appearance gave no suggestion of injury to the nerve at any point; hence he was forced to deduce simple contusion complicated by a profound state of stupor.

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[1] Basset, *Rev. de chir.*, April and May 1916.

Cases of this nature, where in spite of obvious clinical signs and greatly changed electrical reactions, an operation reveals only lesions apparently inadequate to explain these phenomena, are extremely rare in our experience. It seems likely that neuritis, in the inflammatory sense of the word, is a factor which affects the electrical reactions more often than is supposed.

On the other hand, cases have been described where, in spite of extensive or severe compression, giving rise, previous to operation, to serious organic disorders, muscular atrophy, etc., excitability was found to be merely diminished, or even unaltered; in explanation it was suggested that the nerve might acquire a kind of tolerance or power of adaptation to compression.

This absence of changes in the electrical reactions in a case of evident compression with trophic or motor disturbance, should, however, be regarded as an exceptional occurrence. When it does exist, it should be remembered that preservation of the electrical reactions may possibly be accounted for by some anastomosis or abnormal innervation (see p. 151).

There is really, however, no question of tolerance in regard to electrical reactions.

Tolerance or adaptation, in as far as the clinical signs are concerned, may undeniably occur. Ombrédanne has recently published an undoubtedly authentic case of it.[1] An operation had been necessitated by an entirely different condition, and the radial nerve was found to be involved in a large mass of scar tissue. There had been no functional disturbance to suggest a compression of the nerve.

According to Ombrédanne, cases of this kind, where considerable lesions give rise to no disorder of function, are decidedly rare.

Our experience, however, in carrying out numerous electrical examinations for the purposes of Medical Boards, obliges us to take the opposite view. On many occasions we have discovered changes in the electrical reactions, indicating a more or less severe nerve lesion, in patients with no sign of motor or trophic disorders. Cases in which some anomaly in the muscular reactions, as revealed by electrical methods, indicates a compression not clinically recognised, are still of relatively frequent occurrence.

The Reactions of the Exposed Nerve.—If, before an operation, electrical examination has afforded no definite conclusion, would not electrical testing on the operating-table afford more precise information as to the functional value of the injured nerve ?

This procedure would be unnecessary in a case of " discontinuous " section of a nerve, as Claude has expressed it, where the two cut extremities have become more or less widely separated. It would be equally unnecessary if a structure easily removed were found to be causing compression, such as a band of scar tissue, or an accumulation of blood, etc. On the other hand, in the case of a neuroma, a nerve keloid, or a fibrous connection, a conclusion must be drawn at once, as the surgeon must decide whether the continuity of the nerve is or is not interrupted, whether it is advisable not to interfere, or whether, on the contrary, he should boldly divide and trim it, and oppose and suture the two extremities.

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[1] Ombrédanne, *Soc. chirurgie*, March 28th, 1917.

Electro-diagnosis in an operation wound, formerly practised by Bergonié,[1] was exhaustively studied by Pierre Marie and Meige.[2] The exposed nerve is tested with the faradic current by means of a silver or platinum bipolar electrode (single shocks from a coil of coarse winding). If excitability is preserved, Pierre Marie and Meige advise simply trimming and freeing the nerve from neighbouring adherent or bony structures. If, on the other hand, faradic excitability, both above and below the lesion, is abolished, and at the same time "section or marked flattening with an indurated neuroma occupying the whole thickness of the nerve" is discovered, the small chances of ultimate recovery of function suggest excision of the altered segment and suture of the two ends. A less serious lesion will require only a conservative operation.

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[1] Bergonié and G. de Fleury, "Paralysie radial grave par contusion légère. Electrodiagnostic sur le nerf mis à nu et sous chloroforme. Pronostic défavorable," *Arch. d'Électr. Méd.*, January 1901.

[2] Pierre Marie and Meige, "Électrisation du nerf mis à nu," *Bull. Acad. Méd.*, February 9th, 1915.

The method of P. Marie and Meige, based on the condition of faradic excitability, is therefore to differentiate, in the wound, between partial and complete RD: in the former case, the operation will be merely one of freeing from surrounding structures; in the latter it will depend upon the macroscopic appearance of the nerve lesion.

Léri [1] applied electrical methods in the wound in 72 cases of nerve injury. Of these 72 cases, in 8 he found total "discontinuous" section, and absence of faradic excitability; in 15 "continuous" section, that is to say, with a bridge of cicatricial substance, adherent to the neighbouring tissues. In all these cases, as might be expected, there was no faradic response.

In 24 cases in which the nerve was macroscopically intact, and merely more or less adherent to neighbouring tissues, the faradic response was given in 23.

The faradic response in neuromas, however, is variable, sometimes positive, sometimes negative.

In 16 cases of nodular neuroma centred in the axis of the nerve, 8 showed faradic conductivity, which is absolute proof of the continuity of the axis-cylinders; 8 had lost it. Lastly, of 9 cases of lateral neuroma, in 8 there was no response in the region corresponding to the situation of the neuroma.

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[1] Léri, "L'Électrisation directe des tissus nerveux au cours des interventions pour blessures des nerfs," *Paris Méd.*, June 26th, 1915.

It follows from these observations that for purposes of deciding at the last moment whether a neuroma is permeable by stimuli, the method offers certain advantages.

Similarly, a further purpose would be to verify the results of preliminary transcutaneous testing, and to indicate the exact condition in cases of doubt as to the preservation of excitability or faradic conductivity in the nerve. Lastly, in some cases where pronounced œdema or adiposity interfere with the usual electrical examination, direct testing of the nerve will complete our knowledge of the reactions.

Tinel, however, criticises the procedure on the ground that it may lead to the resection of a nerve in process of spontaneous regeneration. Faradic excitability is absent in the initial stages of repair, and remains so until the power of voluntary movement returns, and consequently in relying on this factor alone there would be considerable danger of resecting motor fibres in which the process of regeneration was far advanced.[1]

The Return of the Electrical Reactions.—There is no doubt that serious cases of paralysis have been known to improve under persevering medical treatment.[2]

Some authors, in anticipation of spontaneous recovery,[1] go so far as deliberately to avoid any attempt at treatment.

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[1] Tinel, *Les blessures des nerfs*, Masson, 1916.

[2] Tinel noted improvement in 60 to 70 per cent. of nerve lesions examined 8 to 10 months after the original injury.

Of the physiotherapeutic treatment, the galvanic bath, from its circulatory effects, the continuous current, from the sclerolytic effects of the negative pole, the value of which one of the present writers has continually emphasised in the past,[2] the introduction of certain anions (iodide of potassium[3]), which is doubtless merely a variation of the processes of electric sclerolysis, and lastly radiotherapy,[4] have to their credit a large proportion of favourable results which should certainly be taken into consideration.

Hitherto, however, the repair of anatomical nerve lesions has more frequently been attempted by surgical methods.

The freeing, by surgical means, of nerves undergoing compression, enclosed in a mass of scar tissue, involved in a callus, or displaced and stretched by a ridge of bone, is generally followed, after a variable interval, by recovery of function. With sutures, however, it is a different matter, to judge by statistics hitherto published. Before the war, it was customary to suture divided nerves as soon as possible, and the operation, being aseptic, was frequently followed by functional recovery a few months later. Gunshot wounds being generally infected, it became necessary to postpone nerve suture, a condition of affairs which was the more unfavourable because the muscles subsequently became the site of more advanced trophic changes.

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[1] Huet mentions a case of spontaneous recovery of a radial paralysis following a fracture of the radius. An operation was about to be undertaken when in three days a remarkable improvement occurred.

[2] Zimmern and Cottenot, "La sclérolyse électrique," *Presse Médicale*, June 25th, 1910.

[3] Chiray and Bourguignon, "L'ionisation des cicatrices de guerre," *Presse Méd.*, August 3rd, 1916.

[4] Hesnard, "Le traitement local et la radiothérapie locale des blessures des troncs nerveux," *Arch. d'Élect. Méd.*, January 1916.

In spite of this, good results have been obtained in a number of cases, though always after an interval of many months.

It is true that cases of almost immediate motor recovery have been recorded (by Cazamian[1] in France, and by Lœwenstein[2] in Germany, etc.). These results could scarcely have been hoped for, since it is known that after section of a nerve, the peripheral end degenerates throughout its entire length, and direct regeneration is only possible when the axons of the proximal have penetrated the peripheral end.

Nerves possess a definite individuality (Dumas) from the point of view of the rapidity and completeness of their functional restoration. The progress of the regeneration varies even with the height of the lesion, for the reason that the axis-cylinders have a greater or lesser distance to traverse. The radial is the nerve in which the process of repair is most rapid; function is sometimes recovered in four or five months. The ulnar nerve regenerates slowly. The internal popliteal and the median are the most deceptive in their results.

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[1] Cazamian, " Sur trois cas de suture nerveuse suivie de rétablissement des fonctions du nerf," *Soc. chirurgie*, November 24th, 1915.

[2] Lœwenstein (*Münch. Med. Woch.*, 1916, No. 39) describes a suture of the radial nerve performed fifteen days after its division. On the tenth day after the operation, the power of movement was completely recovered ; on the thirty-seventh day the electrical reactions were normal.

Lastly it should be remembered that the electrical signs of regeneration appear more rapidly in patients who have undergone physiotherapeutic treatment immediately after suture.

It is usually said, on the authority of Duchenne (of Boulogne) that in nerve regeneration, the power of voluntary movement is recovered some time before the electrical reactions reappear.

Stated in this way, the proposition is correct, but incomplete.

Duchenne actually wrote: "I should say at this point that in my experience electro-muscular contractility has always returned, but not completely, and always at a shorter or longer interval after the recovery of voluntary movements and muscular nutrition."[1]

We know now that *the return of faradic excitability of the muscle* is normally *preceded by the return of the faradic and galvanic excitability of the nerve.* "At a certain stage in regeneration," writes Erb,[2] "the faradic excitability of the muscle reappears and gradually approaches the normal. *This usually occurs*

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[1] Duchenne (of Boulogne), *Électris. local.*, éd. 1872, p. 372.

[2] Erb, *Traité d'Électrothérapie.* Trad. Rueff., 1884.

rather later than in the case of the nerve, and is also separated by a rather longer interval from the recovery of voluntary movement."

In the nerve, excitability almost invariably returns later than the normal tone, the sensitiveness of the muscle to the faradic current,[1] and even voluntary movement. The nerve first becomes excitable by the galvanic current, and some time afterwards by the faradic current (single shocks); stimulation produces a brisk contraction in the muscles which it supplies.

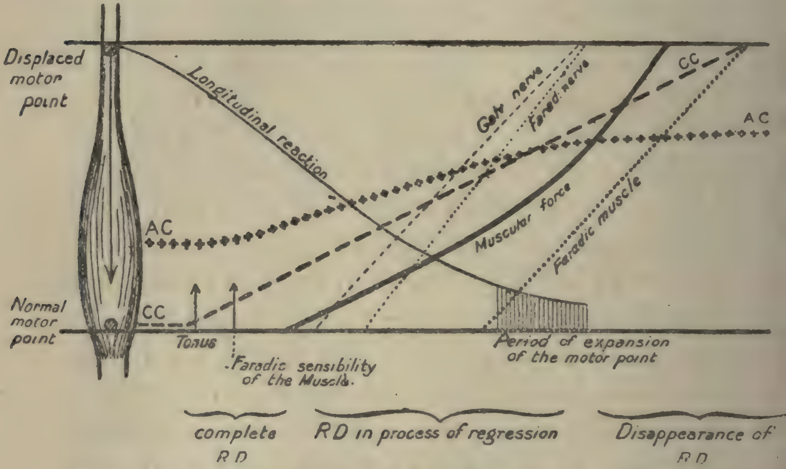

FIG. 41.—Diagram of regressive changes in the electrical reactions (Chartier).

The testing of the nerve should not be confined solely to points below the lesion; early reactions may be given by stimulation *above* the point of division or compression.

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[1] Duchenne (Électris. localisée, *passim*) writes: "The faradisation of a nerve or muscle in the normal state always produces a contraction and a sensation. A nerve lesion must be severe to cause complete loss of sensibility in a muscle. . . . The return of electrical sensibility in the muscles is a favourable sign."

During experiments on the frog at a very early date, Erb observed that injured nerves in process of regeneration regained conductivity before excitability, the nerve being inexcitable below the lesion but excitable above it.[1]

Claude, Vigouroux, and Dumas,[2] record two cases of compression by sclerosis in which they tested the reactions directly during an operation, and found that the excitability of the nerve, which was abolished above the seat of compression, had reappeared some minutes after the nerve was freed from the fibrous tissue enclosing it.

Cottenot and Reinhold have shown that in some cases, when the nerve is still inexcitable below the lesion, a contractile response may be obtained by stimulation above the lesion with the faradic (single shocks) or with the galvanic current. In a case in which excitability had returned in both segments of the injured nerve, they obtained a muscular response with a weaker current in the upper segment than in the lower. According to Cottenot and Reinhold, the return of electrical conductivity in the segment of the nerve above the lesion is always a favourable sign.[3]

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[1] Erb, *Arch. f. klin. Med.*, 1868, p. 535.

[2] Claude, Vigouroux, and Dumas, "Cent cas de lésions traumatiques des nerfs des membres," *Presse Médicale*, March 4th, 1915.

[3] Cottenot and Reinhold, "Une réaction particulière observée au cours de la régénération des nerfs," *Bull. Acad. Méd.*, March 28th, 1915.

In the muscle, faradic excitability reappears after a variable length of time; it is usually subsequent to the return of excitability in the nerve, and always remains less than the normal. Galvanic excitability tends to reappear at the motor point, while the longitudinal reaction diminishes. Hence hypo-excitability becomes less marked. It is not until some time later that the qualitative changes begin to disappear; the contraction gradually becomes more rapid, polar inversion is slowly converted into polar equality, and so on. These changes occur in a definite order which varies with the nerve concerned. The recovery of function coincides with the penetration, by the axis-cylinders, of the degenerated nerve, and consequently the first muscles to return to the normal state are those nearest to the lesion, the process of repair spreading farther and farther until the most distally situated muscles are reached, a clinical picture similar to that of dissociated paralyses.

For a long time after the recovery of voluntary movement, antecedent RD may be detected, as much by a slowness of the contraction or a persisting anomaly in the formula as by faradic and galvanic hypo-excitability.

The delay in the regressive changes of the electrical reactions after the recovery of voluntary movement is sometimes such that the latter may have been complete for several weeks or months without any improvement in the electrical reactions. We have recently seen a case of this kind (radial paralysis) which certainly exhibited incompatible features, the recovery of function being complete, and the muscular development normal, with which was combined absolute inexcitability.

Exceptions to Duchenne's law are not unknown.

Doubtless a certain number may readily be explained by functional disorders, that is to say by the co-existence of a hysterical paralysis. It must, in fact, be remembered that in organic paralyses, anatomical repair may not be followed by recovery of function.

These cases, however, usually exhibit re-development of the muscles from the atrophied condition, a process consistent with the return of the electrical reactions, and equally in contrast with the absence of voluntary movement.

The cases described by Pierre Marie and Mme. Benisty [1] in which faradic excitability or early signs of it reappeared several weeks or months before the return of voluntary movement, are less simply explained. It follows from the observations of these authors that excitability does not always reappear earlier in the nerve than in the muscle. They even discovered cases in which the faradic excitability of the muscles underwent daily variations, disappearing one day to return some days later, and sometimes even disappearing exactly at the time when voluntary movement was recovered.

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[1] Pierre Marie et Mme. Ath. Bénisty, "Du retour de la contractilité faradique avant le rétablissement de la motilité voluntaire dans les muscles paralysés, à la suite des lésions des nerfs périphériques," *Soc. de Neur.*, April 15th, 1915, and May 6th, 1915.

These contradictory phenomena are partly explained by a difference in technique, for instead of using, like Duchenne, the current from a coil of coarse winding, in the form of a tetanising current, these authors employed single shocks from a coil of fine winding, with the maximum intensity (full approximation). Now single shocks are usually more effective, in the pathological condition, than the tetanising current. Dubois,[1] indeed, went as far as to say that he had never seen a single case of complete RD in which single faradic shocks of sufficient intensity were absolutely without effect.

It is obvious that with the diversity of faradic apparatus in use in laboratories, the results of examinations cannot be consistent. In one case the break wave may be of such a form as to be capable of exciting degenerated muscle; in another, it may be too short to cause contraction.

When the faradic excitability of the muscle is tested exclusively with a coil of coarse winding and the tetanising current, it will always be found that Duchenne's law holds good.

The early return of faradic excitability in the muscle, as investigated by this method, has generally appeared to us to be an unfavourable sign.

On the other hand, we regard as a favourable indication the return of faradic excitability (single shocks) or galvanic excitability in the nerve, where this is not preceded by the recovery of voluntary movement.

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[1] Dubois, *loc. cit.*

Earlier indications are afforded by galvanic testing of the muscles. Bourguignon [1] is of the opinion that in cases of nerve suture, slight changes in the galvanic reactions of the muscle usually precede the recovery of motor function (diminution of longitudinal excitability, and lessening of hypo-excitability with galvanotonus, either at both poles or with the positive pole). Similarly a more brisk galvanic contraction, but slight and lacking amplitude, is a bad sign, the amplitude of the contraction being a function of the number of healthy fibres, and its weakness an indication that a number are degenerated.

The evolution of the galvanic reactions of the muscle could obviously be followed very closely by frequently repeated examinations. Hitherto, however, very few electrologists have undertaken an investigation which depends on the recognition of very slight differences, and in which, therefore, the personal equation tends to play a considerable part.

SOME PECULIARITIES OF DIFFERENT NERVES IN ELECTRO-DIAGNOSIS

Facial Nerve.—Direct injury of the facial nerve by a projectile is rare as compared with injuries of the nerves of the limbs. Tinel does not refer to it in his monograph. We have, however, met a certain number of cases.

When the wound involves one of the terminal branches, either in the parotid or beyond, the nerve is usually severed, and the branch in question exhibits complete RD.

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[1] Bourguignon, *in* Chiray et Roger, *Soc. Méd. Hop.*, December 8th, 1916.

In other cases the nerve is injured by the projectile in its passage through the temporal bone. This kind of injury may give rise either to limited or to complete facial paralysis. The affected region usually exhibits complete RD.

Facial paralysis is also one of the symptoms of fractures of the skull. The trunk of the nerve is then injured either in the canal in the petrous portion of the temporal bone, or higher up. The electrical reactions usually exhibit the characters of complete RD towards the end of the second week.

The appearance of partial RD following injuries of the skull, is not, however, necessarily a favourable sign, as in facial paralysis *a frigore*. It should always be remembered that an effusion of blood or an organised exudate may cause progressive and permanent compression. Partial RD in such a case is therefore merely an early stage of complete RD, and an equally unfavourable sign.

Pontine Syndromes.—Fractures of the skull and concussions may give rise, doubtless by hæmorrhage in the region of the pons, to Millard-Gubler's syndrome, in which flaccid hemiplegia affects the face on the side of the lesion and the limbs of the opposite side (crossed hemiplegia).

The word hemiplegia would seem at first to suggest that RD would not be present. In this variety of hemiplegia, however, the facial paralysis has a different anatomical cause from that producing the paralysis of the limbs: it should be observed that it involves the upper division of the facial nerve as well as the lower. Since the radicular fibres or nucleus of the facial are affected, it may be classed among the paralyses of the peripheral motor neurone, *i.e.* amongst those in which RD may develop.

The reaction of degeneration may be complete or partial, but here again partial RD is not a favourable sign as in facial paralysis *a frigore*. The prognosis in the lesion of the facial is further of little importance in comparison with the general prognosis.

Radial Nerve.—Of all paralyses of peripheral nerves, radial paralyses have been of most frequent occurrence during the course of the war.

The distribution of RD among the extensor muscles indicates the site of the lesion. The triceps is rarely affected in lesions of the radial alone; paralysis of this muscle is usually associated with paralysis of the deltoid (lesions of the posterior cord of the brachial plexus). The commonest seat of injury to the radial nerve is the middle third of the arm, whether the cause be direct injury by a bullet or shell fragment, or a fracture of the humerus. In this case the supinator longus is included in the group of muscles paralysed. At the same time, it may remain unaffected in radial paralyses following fractures of the lower extremity of the humerus. The extensor carpi radialis longus and brevior may also be unaffected.

Dissociated paralyses amongst the extensors are rare.

In paralyses by compression (compression by a callus of the humerus, crutch paralysis, paralysis following a dislocation of the shoulder, etc.) RD may be partial or total. Its degree varies with the nature, degree, and duration of the compression. Marked RD is a grave sign, particularly when accompanied by advanced muscular atrophy. According to Pierre Marie and Mme. Bénisty, the radial is the nerve which loses its faradic excitability most readily; the electrical reactions may be absent even with a slight anatomical lesion.

It has always seemed to us that modifications of the electrical excitability, whether qualitative or quantitative, can be detected most readily in the extensor carpi ulnaris. In cases of doubt as to whether the contractions are slow, it is a good plan to test at the motor point and point of junction of tendon and muscle.

In traumatic radial paralyses which recover, the process of repair not infrequently fails to include the extensor pollicis longus, both as regards voluntary movements and electrical reactions.

Cases of slight radial paralysis occur, without marked or even in any way evident changes in the electrical reactions, as evidenced by unipolar faradisation of the postero-superior part of the forearm. Stimuli spread by diffusion to the flexors, and the action of the latter overcomes that of the extensors, of which the excitability is less (Babinski).

Median Nerve.—Paralyses of the median are not infrequently dissociated: a lesion of the nerve in the arm or at the bend of the elbow may produce a paralysis of the flexors only, leaving the pronator teres and the palmaris longus and brevis almost unaffected. In Tinel's view, this dissociation is not absolutely diagnostic of a partial lesion, since the median receives a branch from the musculo-cutaneous in the arm.

A lesion of the median in the forearm is generally evidenced by partial or total RD in the muscles of the thenar eminence. Here the abductor pollicis brevis must be made the standard, since the deep portion of the flexor brevis is supplied by the ulnar nerve, and may consequently respond to the faradic current by reactions suggesting only partial RD, in cases where the median is seriously injured.

The regeneration of the median is always slow; the abductor brevis is the last muscle to recover, and its function is rarely altogether restored.

Babinski calls attention to the fact that in paralysis of the median, stimulation of the biceps produces a movement of more marked supination than normally, owing to the absence of the antagonistic action of the pronator teres.

In slight median paralyses without appreciable change in the electrical reactions, when bipolar faradisation of the forearm produces flexion, monopolar faradisation by application of an electrode in the antero-inferior part of the forearm causes a paradoxical extension of the hand, owing to diffusion of the current to the extensors, which are relatively more excitable (Babinski).

In testing the muscles of the palmar aspect of the hand, it is well, in order to avoid causing the muscles of the forearm to contract, to place the indifferent electrode on the dorsum of the hand.

Ulnar Nerve.—Complete paralysis of the ulnar may easily be missed in a superficial clinical examination, in cases where simple compression or recent severance is the causative lesion, owing to the fact that numerous other muscles supplement the actions of those controlled by this nerve, with the result that almost all movements of the hand are still possible.[1] The importance of changes in the electrical reactions in such a case is evident.

Discrepancies between clinical facts and the electrical reactions seem fairly common in lesions of the ulnar. Thus in even slight compressions of the nerve in the region of the epitrochlear sulcus, which forms an inextensible canal, complete RD follows in some cases, either throughout the region supplied by the nerve, or more locally, and sometimes only in the interossei and the hypothenar eminence.[2]

In a case of Goldmann's,[3] with a tumour of the elbow involving the ulnar, resection of 5 cm. of this nerve produced neither motor nor sensory paralysis. The electrical reactions were similarly found to be unchanged.

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[1] The exception is that the last two fingers cannot be brought together. Loss of adduction of the little finger is often the only apparent sign of a complete ulnar paralysis. It is the only movement which cannot be effected in any other way (Tinel).

[2] Sicard and Gastaud, "Les névrodocites," *Soc. Méd. Hop.*, November 3rd, 1916.

[3] Goldmann, *Neurol. Zentralbl.*, 1906.

Are cases of this kind to be explained by the anastomotic branch from the median ?

Further, we have particulars of a case of simultaneous paresis of the median and ulnar, which exhibited partial RD only ; no improvement occurring, an operation was performed, and the ulnar was found to be completely severed, with a neuroma at the proximal end of the nerve.

Here again, to explain only partial RD in spite of section of the nerve, is the branch from the median to be regarded as playing some part ?

Brachial Plexus.[1]—The brachial plexus may be injured in wounds or their sequelæ, by a projectile, a hematoma, a fracture, a callus, by the formation of scar-tissue, etc. Paralyses of the brachial plexus may also follow injuries to the shoulder, contusions, a fall on the shoulder, depression of the shoulder in structural collapse, dislocations of the shoulder, and manipulation in reduction.

Three varieties are usually distinguished : an upper radicular type (Duchenne-Erb type) from injury of the Vth and VIth cervical nerves, a lower radicular type (Klumpke's type) from injury of the VIIth and VIIIth cervical and Ist thoracic nerves, and lastly, a total radicular type. Sometimes the paralysis is definitely limited to the region of peripheral distribution corresponding to these nerves, and sometimes, on the other hand, the distribution of motor disorders, and more particularly an electrical examination, go to show that the actual distribution of radicular paralyses varies more or less from these fundamental types ; paralyses are not definitely localised in the upper and lower regions, and in other cases the two regions, though simultaneously affected, are affected in different degrees.

The electrical reactions usually develop into complete or partial RD ; very occasionally only faradic and galvanic hypo-excitability appear.

The distribution of RD indicates the radicular region concerned more precisely than is possible by clinical observation, since an investigation of the power of voluntary movement of certain muscles (infraspinatus, teres major, etc.) is almost impossible, and further, on the borders of this region it is not unusual to detect traces of RD in muscles which appear functionally unimpaired.

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[1] Cf. A. Zimmern, "L'électrodiagnostic dans les paralysies radiculaires du plexus brachial," *Paris médical*, 1911.

The problem of the exact localisation of the lesion is extremely complex. Thus in a case of upper radicular paralysis there may be some doubt as to whether the causative lesion is intra- or extra-vertebral. In such cases the reactions of the serratus magnus may give valuable indications. The nerve to the serratus magnus is given off, almost constantly, at the points of exit of the cervical nerves from the intervertebral foramina. The existence of changes in its reactions would therefore indicate a lesion situated higher up. Suture being impossible unless the lesion is extra-vertebral, the importance of testing the reactions of the serratus magnus will readily be understood.

When RD exhibits a complex peripheral distribution, corresponding neither to the peripheral distribution of the terminal nerves nor to the actual radicular distribution, and when the oculo-pupillary syndrome, indicating a lesion near the intervertebral foramen for the first thoracic nerve, is also absent, a lesion in the network of the plexus is suggested. A definite conclusion, however, cannot be drawn, as several solutions are possible ; an unequal distribution of RD may, in fact, be observed when there are co-existing lesions of several roots.

In spite of these difficulties, careful testing of every muscle, rigorous classification of the muscles according to the degree of change in the electrical reactions, and finally systematic interpretation, will often enlighten the surgeon as to the probable seat of the causative lesion.

The peripheral distribution of the five roots forming the brachial plexus will be found tabulated on page 149 : we have only included muscles supplied by terminal or collateral branches which, owing to their superficial situation, are directly and easily accessible to the testing electrode.

Prognosis.—It may be stated as a principle that any traumatic radicular paralysis accompanied by RD, or even by partial RD, is always grave.

Only radicular paralyses unaccompanied by RD suggest a favourable prognosis or can rightly be regarded as benign. In these cases rapid return of function is the rule, and recovery is almost always complete.

The exceptionally grave prognosis in the case of partial RD and the frequent occurrence of forms exhibiting the syndrome of degeneration, give radicular paralysis a particularly grave character unparalleled in affections of the same order. As Huet has pointed out, it would appear that the prognosis in traumatic paralyses of the brachial plexus is more unfavourable for a lesion of

SPINAL ROOTS.	DISTRIBUTION OF THE NERVES						
	Musculo-cutaneous.	Circumflex.	Collaterals.	Collaterals.	Radial.	Median.	Ulnar.
Vth Cervical.	Biceps. Brachialis anticus.	Deltoid. Teres minor.	Rhomboids.	Infraspinatus. Teres major. Pectoralis major. Serratus magnus.			
VIth Cervical	Biceps. Brachialis anticus.	Deltoid. Teres minor.		Infraspinatus. Teres major. Pectoralis major. Serratus magnus.	Supinator longus. Triceps (?).		
VIIth Cervical.				Teres major. Pectoralis major. Serratus magnus. Latissimus dorsi.	Triceps. Extensors. Ext. carpi ulnaris. Ext. Carpi. Rad. Long., and Brev.	Pronator Teres. Palmaris longus. Flexors. Thenar Eminence.	
VIIIth Cervical.				Pectoralis major. Latissimus dorsi.	Triceps.	Flexors.	Flexor carpi ulnaris. Hypothenar. Interossei. Adductor pollicis.
1st Thoracic.				Pectoralis major.		Flexors.	Flexor carpi ulnaris.

given severity, than in traumatic paralyses, as a whole, of peripheral nerves. "The course of radicular paralyses is almost that of myelitis." The peculiar gravity of radicular paralyses is undoubtedly explained by the extension of the injury to the cord, by the more or less severe effects of the causative lesion on the motor cells (traction, concussion, or compression). As Mme. Déjerine has shown, the lesion in lower radicular paralyses may consist in a tearing away of the nerve roots from their points of origin in the cord.

In cases progressing favourably, the circumflex and musculocutaneous nerves are the first to regenerate, followed by the radial, then the ulnar, and finally the median. RD undergoes regressive evolution, and after 5, 6, or 8 months the patient ends by recovering the use of his limb fairly completely, but recovery is more usually only partial, and the functions of the upper limb remain very seriously impaired.

Sciatic Nerve.—Lesions of the trunk of the sciatic are not infrequently dissociated: the level of the bifurcation of the nerve is extremely variable, and is sometimes even at the point of exit from the greater sciatic foramen. The size of the nerve trunk is such that some of its fibres may be injured to the exclusion of the rest, and it is not uncommon to see a complete paralysis of the region supplied by the nerve, subsequently persisting in the region supplied by one of the branches, but exhibiting signs of progressive regeneration in the other.

The external popliteal in its course round the head of the fibula is equally vulnerable by external traumatic agents (projectiles) and by internal agents (fracture of the head of the fibula, or a callus, etc.). A frequent cause of permanent compression liable to produce more or less complete paralysis, and often unrecognised or even attributed to hysteria or simulation, is strangulation by the tibialis anticus or peronei. This form of paresis is almost invariably characterised by partial RD.

SOME ANASTOMOSES AND MIXED OR ABNORMAL INNERVATIONS OF IMPORTANCE IN EXPLAINING THE COEXISTENCE OF CERTAIN VOLUNTARY MOVEMENTS WITH ABNORMAL ELECTRICAL REACTIONS

Mixed Innervations.—The flexor digitorum profundus is supplied both by the median and the ulnar.

The flexor pollicis brevis consists of two parts: the superficial part receives a branch from the median, and the deep part is innervated by the ulnar.

The rhomboids are supplied by a branch from the cervical and a branch from the brachial plexus.

Abnormal Innervations.—The flexor carpi ulnaris is sometimes supplied by the median. The adductor pollicis is sometimes supplied by the median (Froment). The abductor pollicis brevis sometimes receives fibres from the superficial branch of the radial (Lejars).

Brooks has described cases in which the deep branch of the ulnar supplied the flexor pollicis brevis, the opponens pollicis, and the abductor pollicis brevis.

The musculo-cutaneous occasionally takes the place of the median in supplying the pronator teres.

In a case of Turner, the ulnar supplied neither the flexor profundus nor the flexor carpi ulnaris, but sent a branch to the flexor sublimis.

Anastomoses.—*Musculo-cutaneous-median.* Situated in the middle part of the arm; represents a part of the plexus which would complete the distribution of the radicular fibres between the median and the musculo-cutaneous.

Median-ulnar.—(*a*) in the arm: a branch passing behind the brachial artery (Villar); (*b*) in the forearm: a branch consisting of two or more filaments passing in front of the flexor profundus and deep to the flexor carpi ulnaris; its function appears to be to complete the double innervation of the flexor profundus by the median and ulnar.

II. LESIONS OF THE CORD

Cases of compression and partial or complete severance of the spinal cord, whether by the direct action of a projectile or complicating a fracture of the vertebral column, are of frequent occurrence in war pathology.

The changes in the electrical reactions which accompany these lesions principally affect the lumbar and sacral regions, where they may be unilateral or bilateral, symmetrical or diffuse, systematic or dissociated. In lesions situated higher up they may also develop in the upper limbs. In one case of section of the cord, Bastian detected no changes in the electrical reactions. The majority of authors, however, who have had occasion to study cases of this kind, describe very marked hypo-excitability to both forms of current, with or without slowing of the galvanic contraction, and often even absolute inexcitability (Schultze, Pfeiffer) in the whole of the region involved in the paraplegia. Marked hypo-excitability seems to have been a more constant feature than RD itself. According to Brun,[1] this hypo-excitability is only apparent, being due to presence of considerable œdema.

Pierre Marie and Foix regard the changes in the electrical reactions as resulting, at least in a number of cases, from a neuritis in the affected limbs, usually most marked in the external popliteal nerve.[2]

A study of the alterations in electrical reactions is of no great value in direct injuries to the cord, unless, as in some cases, its purpose is to determine the level of the lesion, and hence to verify a radiographic examination.

Much more valuable information may be gained by testing the reactions in contusions of the cord (falls, burying, or the impact of a projectile), and concussions by shell or mine explosion.

Paralyses following injuries of this nature are often accompanied by a slight or marked degree of RD, attributable to lesions of the anterior horns (hematomyelia).

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[1] Brun, *Arch. f. psych.*, 1893.

[2] P. Marie and Foix, *Soc. de neurol.*, March 2nd, 1916.

In severe cases, hematomyelia is clearly evidenced by a number of characteristic clinical symptoms (paraplegia and syringomyelic dissociation), but in the less severe forms, the discovery of abnormal reactions ranks, with lumbar puncture, as of the greatest diagnostic value.

In mild cases, where the supplementary action of other muscles is liable to mask the paralysis, one muscle or a group of muscles on one side or both, usually in the region of distribution of the sciatic, and more particularly in that of the external popliteal, will be found to exhibit either a mere diminution of excitability or partial or complete RD.

Since changes in the anterior horns may affect the grey matter at different levels, the corresponding changes in the electrical reactions may appear in a number of widely separated muscles of altogether different groups.

The degree of RD is usually a sure indication of the prognosis : cases where only a limited number of muscles are involved, and RD is only partial, usually recover without difficulty.

III. COMBINED HYSTERICAL AND ORGANIC DISORDERS

The clinical symptoms in wound cases not infrequently fail to correspond with the classical descriptions of peripheral paralyses. When a nerve lesion is complicated by direct injury to muscles, or when joints, and particularly small joints, have become stiff owing to prolonged immobilisation, the loss of function will appear greater than the nerve lesion itself can account for. It will readily be understood that electrical examination is of great value in such cases in determining the extent of the part played by the nerve lesion in producing these symptoms.

In other cases the simultaneous injury of several nerves, each giving rise to a different paralysis, will render the clinical diagnosis extremely laborious. It seems hardly necessary to emphasise the value of an electrical examination, not merely from the diagnostic point of view, but also in determining the degree of severity and probable nature of the lesion in each case.

The same is the case with superimposed psychical paralyses, which sometimes so strangely transform the clinical picture in an organic paralysis.

It is not unusual, for instance, to find a patient with an organic lesion of the ulnar and median nerves unable to raise the wrist or extend the fingers.

Sometimes these additional paralyses are immediate, following directly on the injury; sometimes they develop gradually, particularly when the patient is kept at rest for a long time, and care has not been taken to correct the more powerful action of antagonising muscles by means of suitable elastic appliances.

In every case, the absence of pathological reactions, contrasting with the severity of functional disorder observed clinically, will indicate the real cause of the paralysis. It should not, however, be forgotten that the diagnosis can only be made with certainty after an interval of about ten days, that is to say, until sufficient time has been given for early anomalies of the reactions to develop.

IV. REFLEX PARALYSES AND CONTRACTURES (PHYSIOPATHIC DISORDERS)

The preservation of the normal electrical reactions in functional disorders of a hysterical nature is now an established fact, and consequently a factor of the greatest importance in diagnosis.

Certain cases have, however, been described in the past, rather as curiosities, of functional disorders which gave rise, either merely to a slowed contraction or to partial RD, and such observations were regarded with incredulity by those who had not actually observed these phenomena themselves. Possibly these paradoxical reactions may be explained by a study of the physiopathic disorders appearing during the present war.

It will be remembered that Babinski and Froment have established the relationship between the disorders which they describe as paralyses (and contractures) of a reflex nature, and the amyotrophic reflex paralyses first studied by Charcot and Vulpian.

The variety known as articular or abarticular, which follows injuries or inflammations of joints, is the commonest type. It is well known to medical electrologists, possibly more so than to surgeons and neurologists, since the patient usually comes direct to an electrologist to be relieved of this persistent functional trouble.

Quantitative changes in excitability are the only abnormalities in electrical reactions occurring in these articular amyotrophies. The reaction of degeneration is never present. Faradic and galvanic excitability are more or less diminished, in some cases preceded by a stage of faradic hyper-excitability (Mally). Not infrequently excitability to the galvanic current is more markedly reduced than to the faradic. Evidence of this diminution of excitability is shown, not merely by a higher threshold, but by reduced amplitude of the maximal contraction.

According to Mally, the prognosis in reflex articular atrophy depends on the extent to which the galvanic excitability of the muscle is diminished, an increase in galvanic excitability being a favourable sign.

We should also emphasise, in passing, the importance of testing the electrical reactions in the particular type of paralysis following injuries to the shoulder (fractures of the neck of the humerus, dislocations, etc.). These are often diagnosed as reflex atrophy. The presence of the reaction of degeneration will indicate their true causation—neuritis of the circumflex nerve, compression, or more or less extensive lesions of the spinal nerve roots.

Since the beginning of the war, physicians have often been struck by the number of atypical disorders, exhibiting neither the features of hysterical manifestations nor those of organic nerve lesions.

Owing largely to their novelty, these conditions have been investigated by a number of workers, and a particularly good description of them has been given by Babinski and Froment.

Amongst their distinctive features, together with *mechanical hyper-excitability of the muscles and the slowness of the contraction excited by percussion*, Babinski and Froment describe two electrical signs—*slight hyper-excitability or hypo-excitability*, and *premature fusion of contractions.*[1]

The quantitative abnormalities on faradic stimulation are never pronounced; sometimes the difference between the healthy and affected sides is so slight as to suggest that quantitative changes have not occurred.

When they are evident, steps should be taken to show that they are not merely apparent, and due to increased or reduced skin resistance on the affected side. We have often observed a marked alteration in skin resistance in reflex paralyses and contractures.

We have already indicated [2] that cold alone may modify the character of the galvanic contraction, and that its action is indirect, through the circulatory disturbance which it produces. It would therefore be reasonable to expect a slowed contraction in reflex disorders accompanied by hypothermia and vaso-motor paralysis. We are inclined to go further in explaining the phenomenon, and to suggest that it may also be produced by asphyxia, CO_2 poisoning, a sluggish circulation, and imperfect exchange between the blood and the tissues.

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[1] Babinski and Froment, *Hystérie. Pithiatisme et troubles nerveux d'ordre réflexe.* Paris, Masson, 1917.

[2] See p. 77, Sources of error in deducing RD.

The changes in excitability and the character of the muscular contraction can be determined most readily in the small muscles of the hand (thenar and hypothenar eminences and interossei).

Faradic excitability is sometimes slightly increased, sometimes slightly diminished. The contractions produced by single shocks may appear fairly brisk, but it will often be found that the number of stimuli per second which will produce tetanus is lower than in the case of a healthy muscle of approximately equal size. This phenomenon of premature fusion of contractions (Babinski and Froment) is therefore indirectly indicative of slow faradic contraction.

With the galvanic current, excitability may be increased or diminished in different cases; the contraction is sometimes markedly and sometimes only slightly slower than the normal.

Artificial cooling of a limb produces slowing of the contraction; if then it be adequately warmed, the contraction becomes more rapid and finally normal.

All these abnormal phenomena originate in disturbances of temperature, and disappear with them. Increased rapidity of contraction on warming will always distinguish these reactions from the reaction of degeneration.

V. ISCHÆMIC PARALYSES

Local anæmia may produce marked changes in the electrical reactions. These were demonstrated by Marey, who observed slowing of the contraction after ligature of the artery supplying the muscle.

The effects in man are variable ; sometimes typical RD appears, but it is commoner to find a remarkable degree of hypo-excitability, amounting sometimes to inexcitability, with or without polar inversion. This inexcitability of the muscles is often earlier and more pronounced than the inexcitability of the nerve supplying them (Tinel).

The electrical reactions are also more or less markedly altered in certain pathological conditions of common occurrence during the present war, which have been compared with Volkmann's ischæmic contracture (pain, œdema, hypothermia, wooden hardening of the muscles, fibrous retractions).

Any condition intermediate between simple hypo-excitability and complete RD may be present. Further, the distribution of these effects is extremely irregular ; one muscle may exhibit complete RD and its immediate neighbour simple hypo-excitability. In the small muscles of the hand, testing the reactions is an easy matter ; in the flexors, however, retraction and fibrosis may render it entirely impossible.

The anomalous electrical reactions in Volkmann's syndrome are sometimes attributed to ischæmia, but it seems much more logical to attribute them either to a neuritic process connected with the muscular changes, or simply to strangulation of the nerve fibres in masses of fibrous tissue.

VI. PARALYSES OF TOXIC AND INFECTIVE ORIGIN. NON-TRAUMATIC FORMS OF NEURITIS, MYELITIS, ETC.

Paralyses due to infective or toxic neuritis are as common in military ambulance stations and hospitals as in civil practice. We shall therefore describe the essential features of electro-diagnosis in each type of case.

Peripheral Facial Paralysis.—Facial paralysis *a frigore,* an extremely common condition, may assume either of two forms, the benign or the severe. Although the severe form may not be followed by permanent paralysis, its effects will render the patient unfit for further service, particularly when lachrymation interferes with vision, and inability to close the eye leaves the conjunctiva unprotected.

It is impossible to distinguish clinically between the slight and severe forms. There is nothing in the clinical signs of facial paralysis *a frigore* to suggest whether the lesion is severe or otherwise, or its probable duration.

Electrical methods are the only means of obtaining this information. Erb, in fact, showed that from the results of an electrical examination, any given case could be classed in one of the following categories—

mild form, intermediate form, and severe form, the course and prognosis being different in each case.

1. In the *mild form* of facial paralysis, the electrical reactions remain normal. The outlook in these cases is always favourable, and recovery usually takes place within *three weeks.* Unfortunately a relatively small proportion of cases assume this form.

2. In the forms exhibiting RD, the quantitative and qualitative changes preceding the appearance of definite RD (complete or partial) usually develop during the first two weeks. These changes are only slight for some days, and, as a rule, the abnormal reactions only begin to take definite shape towards the end of the first week; the development of RD is not complete until *the thirteenth or fourteenth day.*

It will therefore be obvious that an examination at an earlier period, finding the muscles in the stage of diminishing faradic excitability, will furnish no definite indication as to the form of the paralysis. Hence the rule that *no conclusions* as to the severity or prognosis in a case of facial paralysis should be drawn *before the end of the second week.*

The essential and remarkable characteristic of the progress of RD in facial paralyses is *prolongation of the stage of galvanic hyper-excitability.*

(*a*) The *intermediate form* is distinguished by the presence of partial RD.

As in the first variety, recovery is usual in the intermediate form, but the process is a longer one. The return of voluntary movement usually begins at about the second month, and *recovery is not complete until three to six months* have elapsed. The electrical reactions remain abnormal for much longer periods, regressive changes beginning long after the power of voluntary movement has been recovered.

(*b*) The *severe form* is characterised by the presence of complete RD.

The prognosis is always unfavourable. Recovery, if it takes place, is very rarely complete. Traces of the paralysis usually persist in the form, either of slight paresis, evident in any change of facial expression, or of a certain asymmetry of the face, often the more evident owing to some slight degree of contracture. The first voluntary movements in these cases usually appear *between the third and the tenth month.*

The recovery of voluntary movements often precedes the return of the electrical reactions to the normal by a long interval, and many months elapse before the muscles regain their faradic excitability. Sometimes the regressive evolution of RD is so slow, and faradic and galvanic excitability remain diminished for so long, that an examination of old facial paralyses which have recovered will often reveal a marked reduction in both forms of excitability and obvious traces of slowed contraction, long after clinical improvement has ceased.

When voluntary movements fail to reappear within ten months, the likelihood of recovery becomes very doubtful.

In incurable cases, a considerably prolonged stage of galvanic hyper-excitability is followed by a period of *progressive hypo-excitability*; the muscular response becomes more and more difficult to elicit by galvanic stimulation. If inversion of the formula has not occurred earlier, it will appear at this stage; at first ACC = KCC, and later the AC contraction becomes greater than the KC.

In many cases *the reaction of exhaustion* will also be detected by galvanic excitation. Repeated galvanic stimuli at short intervals provoke contractions which become progressively weaker.

The incurable forms sometimes end in atrophy of one side of the face, but a much more common result is permanent secondary contracture. This condition only occurs as a sequel to the severe forms exhibiting complete RD. It usually begins to appear during the third or fourth month, and is at first localised in the zygomaticus, extending thence to the levator labii superioris alæque nasi, drawing the labial commissure upwards and outwards. This contracture is sometimes transitory, but more often permanent and intractable. It is an almost invariable sequela in older individuals, and in cases where the general condition is poor at the time of onset.

The following signs indicate the approach of this condition:

1. Spasmodic contractions, occurring either spontaneously, or as the result of artificial stimuli (electrical, or percussion).
2. The rapid recovery of tone when voluntary movement is still impossible.
3. The return of faradic excitability in the muscles in an unusual order (Duchenne).

Duchenne gave the following as the order in which the muscles recover their excitability, in curable forms: buccinator, zygomatici, levator labii superioris alæque nasi, frontalis, orbicularis oculi, and lastly the dilatores nasi.

4. The appearance of certain associated movements: if the patient is told to close his eyes tightly, there is a simultaneous upward and outward movement of the labial commissure and the ala of the nose (Huet). Similarly the palpebral fissure is narrowed when the patient attempts to smile.

The *entire absence of clinical signs* to indicate the severity of a case of facial paralysis, lends additional importance to the results of electrical examination, as we have already seen. Electrical methods, however, although they may determine the diagnosis of a severe form, cannot decide the prognosis. There is nothing in the reactions to indicate beforehand whether the paralysis will pass off or remain permanent, or what degree of contracture is to be expected.

Facial paralysis is common in *infective otitis.* It commonly affects the upper and lower divisions unequally, and is more usually confined to the lower, in which its effects become more or less marked. In all long-standing cases of suppurative otitis there is slight deviation of the labial commissure, the only sign of paresis of the lower division of the facial nerve (Moure).

These pareses or paralyses are usually accompanied by partial RD, and the changes in the reactions are always less marked in the upper part of the face; in total paralyses, this sign is the earliest indication of an improvement.

Otitic facial paralysis with complete RD is generally the result of purulent otitis, mastoid abscess, or caries of the temporal bone; it has also been known to follow surgical trauma in operations on the ear (curetting and trephining of the mastoid, etc.).

Here again complete RD is a serious sign, and indicates an unfavourable course; all facial paralyses accompanied by complete RD are not, however, incurable.

Radial Paralysis. — Common radial paralysis, described as radial paralysis *a frigore*, is most commonly caused by compression and stretching of the nerve (pressure against the edge of a table, or upon the operation table during general anæsthesia, against the back of a chair in individuals who fall asleep in this position, or against the humerus in persons who sleep with the arm bent beneath the head, and so on).

In such cases the nerve is subjected to pressure, but only for a short period, whereas in paralyses by compression (the pressure of a callus or a crutch, etc.), the cause of compression is permanent, progressive, or renewed at intervals indefinitely.

The electrical excitability in radial paralysis *a frigore* undergoes very little change; a slight reduction in the faradic excitability of the muscles, unaccompanied by changes in the galvanic contraction, is the only anomaly liable to occur.

Cases have certainly been reported of radial paralysis from compression during sleep, which were accompanied by signs of RD. These are, properly speaking, exceptions which in no way invalidate the rule, and can be explained on the grounds that the nerve has to some extent absorbed a toxic substance, generally alcohol. Radial paralysis of this type in drinkers certainly occurs much more commonly in a drunken sleep than otherwise.

In common radial paralysis, stimulation above the point of compression, as we have already seen, may suggest false conclusions as to the electrical reactions. The conductivity of the axis-cylinder is often abolished below the point of compression, so that faradic stimulation in the axilla causes no contraction of the extensors of the hand, but may provoke contraction of the triceps, the nerve to which arises above the point of compression.

The existence of faradic excitability may be demonstrated by applying the electrode below this point (Erb).

Radicular Paralyses of the Brachial Plexus.—Radicular paralyses result either from lesions of a surgical nature (stretching, rupture, and compression of the nerve roots) (see p. 147), or from inflammatory changes, or from some toxic cause. The second category includes paralyses due to involvement of the spinal roots in inflammations of the meninges (cerebro-spinal and other forms of meningitis), in addition to primary affections of the nerve roots.

Radiculitis and the radicular paralyses of meningitis are accompanied in their course by complete or partial RD or merely by diminution of excitability to both currents, according to the degree of severity of the lesion. The reactions develop slowly or rapidly, according as the disease is chronic, subacute, or acute. When the abnormal reactions have fully developed, and are found to be definitely characterised in one of the radicular regions of distribution (upper, lower, or both), the limits of this region will not be clearly defined; various muscles are unequally affected, some exhibiting total and others partial RD. Sometimes the topography of the reaction is so irregular that it becomes scarcely recognisable as a radicular distribution.

This is explained by the fact that different roots may be affected unequally.

The distribution of the electrical reactions is not usually sufficient in itself to distinguish between acute poliomyelitis localised in the upper limb segments, and radicular inflammation. In both cases the disease may spread beyond the cervical region, and paralytic disorders, with altered reactions, may be observed in the intercostal muscles and the lower limbs, or on the opposite side.

From the point of view of recovery of function, RD is not as serious a sign in radiculitis as in poliomyelitis.

Disappearance of the signs of inflammation, and good results from treatment in the syphilitic forms, give some hope of recovery; but granting that the radicular lesions are capable of repair, functional recovery is usually imperfect, and never as satisfactory as in peripheral neuritis, for example. It may be stated in a general way that from the point of view of its significance in prognosis, RD in radiculitis stands intermediately between RD in poliomyelitis, and RD in multiple neuritis.

Sciatica.—Sciatica *a frigore*, or from a toxic or infective cause (syphilis, gonorrhœa, tuberculosis, gout, etc.), if accompanied by functional impairment or muscular atrophy, always presents some degree of change in the electrical reactions, which finally may amount to complete RD.

Intermediate between sciatic neuritis with trophic disorders, and simple sciatic neuralgia, are a large number of cases in which the electrical reactions are not customarily tested.

According to Néri, faradic and galvanic excitability are often diminished, more notably in the internal than the external popliteal nerve.[1]

This difference is further shown by diffuse faradic excitation of the sole of the foot, which in certain cases of sciatica causes extension of the toes, instead of producing flexion, as would be the case normally.

Multiple Neuritis.—Infective multiple neuritis (in influenza, typhoid, diphtheria, typhus, leprosy, and beriberi) and toxic multiple neuritis (alcoholic and syphilitic) have been met with frequently in the course of the war.

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[1] V. Néri, "Les petits signes électriques de la sciatique," *Bull. delle Sc. med.*, February 1917.

Except in rapidly curable forms of alcoholic paraplegia and generalised diphtheritic paralysis, RD is the rule in multiple neuritis. In many cases it is even developed earlier than the first manifestations of paralysis.

RD possesses a definite character in multiple neuritis, and accurately reflects the histological changes occurring in the nerves. In the first place, its extent and distribution among a large number of muscles belonging to different groups indicate the multiplicity and widespread distribution of the nervous lesions. Further, its variations in degree from one region to another, or from one muscle to another, reflect the irregularity and diffuse nature of the anatomical process. It usually does, in fact, appear in different degrees; one muscle may exhibit complete RD, and another, supplied by the same nerve, only partial RD, or possibly complete RD with less marked diminution in galvanic excitability. Certain cases show only partial RD, but almost always with the same obvious differences in the extent of diminution of faradic excitability.

It is not unusual in multiple neuritis to find that the extent of the paralysis and the degree of alteration in the electrical reactions are far from corresponding. There may be profound changes in the electrical reactions, complete RD, slow contraction, or absence of faradic excitability, while the muscle responds normally to voluntary impulses.

RD may sometimes be the first indication that neuritis is present. It often happens that no attention is paid to the conditions of weakness in the lower limbs appearing in certain patients, during convalescence from influenza, for instance. Now in cases of this kind, *some traces of RD* will often be detected on electrical examination, and the actual cause of this loss of power will be recognised.

In the same way, a certain difficulty in marching, or a condition of continual fatigue in the lower limbs, should arouse the medical officer's suspicions. An electrical examination will often show traces of RD, which will lead to the discovery of the fact that the patient has been drinking excessively, and incidentally explain the merely subjective symptoms of which he complains.

The degree of severity of multiple neuritis varies, in the first place, with the intensity and the extent of distribution of the anatomical changes in the nerves, and further, with the nature of the underlying cause.

Almost all forms will recover, unless certain of the cranial nerves become involved.

RD is therefore not a peculiarly bad sign in multiple neuritis. A muscle which will only respond to longitudinal stimulation does not undergo atrophy and permanent loss of function, as usually occurs in poliomyelitis.

Alcoholic neuritis more particularly affects the lower limbs, especially the extensor group (quadriceps femoris, extensor digitorum longus, extensor hallucis longus, the peronei, and tibialis anticus). The relative gravity of the prognosis is explained by its long duration, and by the fact that deformity is a common result.

The severity of infective multiple neuritis varies with the degree of RD and the extent of its muscular distribution. In the forms involving a large number of muscles, recovery is always slow, and some of these muscles may remain permanently affected.

Diphtheritic paralysis, a comparatively rare condition, is sometimes accompanied by partial RD (paraplegic forms, and diphtheritic facial paralysis). Complete RD is exceptional. It would therefore appear that the diphtheria toxin has a less severe effect on nerve tissues than other toxic processes.

Multiple neuritis has been observed to follow *frostbite*, and even *gassing*, during the course of the war.

These forms are usually localised to the extensors of the leg, symmetrical, bilateral, and accompanied by partial or complete RD.

According to Tinel, the tibialis anticus is often unaffected, as is the case with the supinator longus in lead poisoning.

Poliomyelitis.—Poliomyelitis in the adult has appeared several times during the war.

As in infantile poliomyelitis, cases unaccompanied by RD, and ending in spontaneous and complete recovery, are extremely rare.

In the vast majority there are profound changes in the electrical reactions.

Poliomyelitis in the adult is usually less systematised than the infantile form. It has a selective action on the lower limb, but its effects are not confined to the antero-external group of leg muscles. It often affects the thigh or the opposite limb simultaneously, and sometimes also the upper limb.

The differential diagnosis from multiple neuritis is often extremely difficult. Clinically, the mode of onset and the presence of sensory disturbance will leave no doubt as to the nature of the disease. From the point of view of electrical reactions, wide distribution and unequal degrees of RD in adjacent muscles suggest multiple neuritis, and a radicular distribution suggests poliomyelitis. Conclusions have frequently to be based on very slight differences.

The prognosis varies with the extent to which the electrical reactions are modified. The presence and persistence of complete RD, with inexcitability at the motor point, indicate severe degenerative changes in the anterior horn cells, and leave little hope of recovery of function.

Partial RD, on the other hand, indicates that the corresponding cells in the grey matter are less completely involved, and although it is to be feared that the muscle will never recover its original strength and development, it will certainly not undergo total atrophy.

VII. THE SEVERANCE OF MUSCLES AND TENDONS

Gunshot wounds frequently involve severance of muscles or tendons, caused either directly by the projectile itself (a fragment of shell) or by suppurative erosion.

It may be of importance to know what tendon or tendons have been cut, and in this connection electrical methods may be of great service.

Assuming a patient to be incapable of performing a certain definite movement, either the electrical reactions of all the muscles will be normal and stimulation will produce normal movements at the joint concerned, which suggests that the paralysis is psychical in origin; or the reactions will be abnormal, with or without RD, which suggests some form of neuritis; or the reactions will be normal, but muscular contraction will produce no movement of flexion or extension, adduction or abduction, and so on, in which case there is every reason to suppose that the tendon is divided.

In such a case it is best to test with the tetanising faradic current, and the electrodes are applied close together over the fleshy part of the muscle, to avoid diffusion of the current. A series of sustained contractions is produced by means of the key. It will then be observed that contraction of the muscle causes no corresponding movement in the part into which its tendon is inserted, whereas stimulation of adjacent muscles produces movement. The belly of the muscle will be felt to swell beneath the touch, but the finger will not detect any movement of the tendon.

During this examination it is essential to place the limb in such a position that a certain amplitude of movement is possible, a precaution recommended by Duchenne (of Boulogne). To facilitate a movement of flexion at any given joint, the limb must be extended, and *vice versa*.

Further, it is often necessary, as Laquerrière observed, to immobolise certain parts: to be sure, for instance, that the terminal phalanx is being flexed, the second must be prevented from moving, or the whole finger will be drawn towards the palm of the hand, which is no evidence as to the condition of the flexor profundus.

The value of an electrical examination in cases of this kind is enhanced by the fact that ability to execute voluntary movements may conceal the presence of a severed tendon. Muscular groups rather than single muscles are employed in voluntary movements;

moreover, a divided muscle is always retracted, and it is almost impossible to detect its contraction by touch.

Unfortunately, the proportion of such cases presenting nothing more than simple division of a tendon is much lower in the case of gunshot wounds than in accidents in civil practice.

Ruptured tendons are often embedded in scar tissue adherent to neighbouring structures, which frequently forms a large fibrous mass from which it is impossible to dissect a muscle or tendon.

CHAPTER IV

ELECTRO-DIAGNOSIS IN DISORDERS OF SENSATION AND THE SPECIAL SENSES

I. Disorders of Cutaneous Sensation

Farado-cutaneous Sensibility and Sensibility to Pain.—We have seen that under certain conditions of technique the faradic current is liable to produce marked sensory effects. Using current from a coil of fine winding (current of tension), and an active electrode of metal rather than a moist electrode, the skin being smeared with a thin film of vaseline, sensibility can be tested much more satisfactorily than by using a needle. In the first place, the fact that the skin is not injured by the process enables the operator to repeat stimuli indefinitely without leaving any trace of them; further, the stimulus may readily be graduated and measured by moving the secondary coil; and finally, stimulation can be carried to such a point that all forms of anæsthesia, other than complete organic anæsthesia, can be overcome by the current. With these advantages the faradic coil is a valuable means of testing sensibility, both from the point of view of affections of the sensory system, and for the purpose of exposing malingerers.

When a metallic electrode (Tripier's or Duchenne's brush) is passed lightly over a limited skin area, with the interrupter vibrating rapidly, and when at the same time the secondary coil is moved slowly up towards the primary, a point is reached (threshold of stimulation), at which the patient begins to experience a slight *tickling* sensation. This is the *farado-cutaneous sensation.* With an increase in the strength of the current, this becomes a tingling sensation, which is at first disagreeable, and gradually becomes definitely *painful.*

The first sensation of tickling and the sensation of definite pain do not, in healthy subjects and for any given region, correspond to a fixed and invariable intensity of current, but usually approximate to an average obtained with a given position of the coils in each faradic apparatus. By noting on the one hand the threshold of farado-cutaneous sensation, and on the other the threshold of painful sensation, two figures are obtained between which is included what is known as the *faradic interval.*

The intensities required are practically equal for corresponding points on opposite sides, but vary with different regions of the skin surface. The sensation produced by a given current in the tips of the fingers, for instance, is well marked, whereas on the outer aspect of the thigh the same current will be scarcely perceptible.

To avoid all possible error due to suggestion, it is essential, in testing to determine the threshold, to repeat the tests several times, and only to accept numerical results (measured in cms. of distance between the coils) which approximately agree.

Faradic Testing in Disorders of Sensibility. — Investigations of *the variations in sensibility in pathological conditions* (Drossdorf, Erb, Boudet de Pâris, etc.) have been directed to estimating, first, the threshold of farado-cutaneous sensation; secondly, the threshold of painful sensation; and lastly, variations of the faradic interval.

Pathological alterations of sensibility to the faradic current occur in organic disease and functional disorders, accompanied usually by sensory changes (neuroses, certain cerebral and spinal affections, tabes, section of the cord, certain forms of neuritis, etc.).

It was natural to suppose that the two stages in faradic sensation, farado-cutaneous sensation and painful sensation, corresponded to two forms of sensibility, sensibility to touch and sensibility to pain, and it was hoped that the intensity of the threshold stimulus would afford an objective measure of *quantitative pathological changes* in these two forms of sensibility. Actually, however, this is not so; as a general rule, the electrical sensibility of the skin varies in the same way as the sensibility to pain.

It is true that there are some exceptions to this rule. In tabes, for instance, as was shown by Erb, farado-cutaneous sensation may be normal, while the sensation of pain cannot be produced even with a current of maximum strength. In this case, the "faradic interval" is equal to infinity ($I = \infty$).

According to Huet, similar changes may be met with in syringomyelia. They also occur in Brown-Séquard's paralysis. Except in these particular cases, variations in the sensibility to pain can be determined more accurately by means of the faradic current than by the usual clinical methods.

Determination of the exact limits of the area affected, objective measurement of changes in sensibility by a more delicate method than that of the needle, and study of the development of a sensory disorder by definite measurements, represent the advantages to the physician of testing sensibility electrically. It is worthy of remark that larger areas of altered sensation will often be detected by this procedure than by other means of investigation.

Hysterical anæsthesia, in addition to its segmental distribution, which can readily be verified by the faradic current, is also characteristic in being only partial. A current of a certain strength will always, in fact, give rise to sensation. This is the basis of the method of psychotherapy, which is now generally recognised as the most effective treatment.

Severance of a nerve should, in theory, produce total faradic anæsthesia, but in general it is only partial unless more than one nerve has been divided; in other words, it is important to take connecting and recurrent branches into consideration.

Testing sensibility may be of great value in the clinical and topographical diagnosis of grave lesions of the cord. As in the case of a divided nerve, faradic testing will indicate the exact point at which the sensory path is interrupted.

In hemiplegia with crossed hemianæsthesia (Brown-Séquard's paralysis), faradic testing will show hyperalgesia on the side of the lesion, and on the opposite side an increase in the faradic interval, constituted by a greatly raised threshold of painful sensation (marked hypoalgesia) and a normal or slightly raised threshold of farado-cutaneous sensibility.

The Galvano-psychical Reflex.—With a very sensitive galvanometer, measuring extremely weak currents and graduated in micro-amperes, it is possible to determine whether certain disorders of sensation and the special senses are functional or organic, through the medium of the galvano-psychical reflex.

Discovered by Féré, and taken up later by Veraguth, this phenomenon has recently been studied in the course of military experience by one of the writers in collaboration with Logre.[1]

If a healthy subject be placed in a galvanic circuit of very low electromotive force (4 to 6 volts), the electrodes, which should be of metal and of considerable surface area, being kept closely in contact with the palms of the hands, any emotion or stimulation of any sensory nerve produces an instantaneous deflection of the galvanometer, indicating a sudden reduction of resistance at the points of contact with the body.

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[1] Zimmern and Logre, *Soc. de Neur.*, June and July 1917.

Any emotions, surprise, attention, or mental processes may give the reflex ; for this reason psychiaters have attempted utilising it as a measure of affectivity.

Sensory stimuli (touch, pricking, and heat), visual stimuli (a magnesium flash, for instance), and auditory stimuli (the ringing of a bell or vibration of a telephone transmitter), will also give rise to the phenomenon. Of the three forms of sensory stimulation, heat seems to be the most reliable and effective, in the sense that it always causes more galvanometric deflection than either of the others.

Certain authors have affirmed that these sensory stimuli do not act directly, but indirectly through an affective element (emotion resulting from perception), which would necessarily imply a psychical connecting link in the galvano-psychical reflex. It appears to us, however, that this affective stage in the reflex path is not essential to the production of the phenomenon, since in the case of hysterical subjects, in whom the reaction is usually pronounced, it would imply subconscious or conscious perception.

In extensive and profound organic anæsthesiæ (division of large nerve trunks without recurrent sensibility, division of the spinal cord, and tabes), if stimulation is carried out unobserved by the patient, to obviate the effects of expectant anticipation, no deflection of the galvanometer will follow. On the other hand, stimulation in a healthy region for purposes of control will produce deflection. In hysteria, the amount of deflection is usually greater than in a healthy subject, and if the sensory disturbance is unilateral, the affected side will give a more pronounced response.

The same applies to the deaf: psychical deafness, or deafness which is largely functional, will always give a more or less marked deflection. In a case where the cause of deafness is organic, and hearing has really been lost, a loud stimulus has no effect, whereas a thermal stimulus applied to the skin of the patient will cause a deflection.

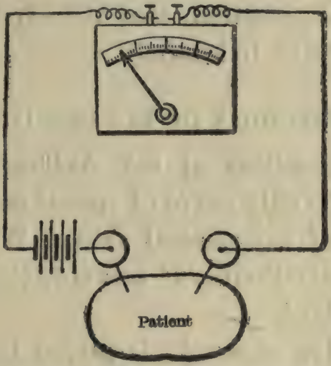

Fig. 42.—Apparatus for the galvano-psychic reflex.

It follows from these observations that the presence or exaggeration of the reflex will in some cases lead to suspicion, and in others prove that the sensory affection concerned is of a functional nature. Absence of the reflex, when satisfactory control tests have been performed in unaffected regions, is absolute proof of an organic lesion and of the genuineness of the case.

The physiological cause of the phenomenon is the sudden expulsion of sweat retained in the bodies of the sweat glands, by contraction of the unstriped muscle surrounding the epithelium of the lumen. The descending path of the reflex is through the sympathetic nerves, which control this momentary excretion. The reflex will obviously not be obtained if the sweat glands are not secreting, or secreting insufficiently, and it will be evident from the circumstances of its causation, that the expulsion of sweat will not continue indefinitely.

II. AFFECTIONS OF THE SPECIAL SENSES

Electrical Reactions of the Auditory Nerve. — The auditory is the only nerve of special sense the testing of which is of any clinical value. We shall merely summarise from Roques's[1] account, the results which may be obtained.

If the active electrode is placed in the external meatus of the ear to be tested, and the passive electrode some distance away, it will be found that the strength of the current can be increased to more than 10 milliamperes without producing a marked auditory sensation at kathodal closing. This sensation is only given with higher intensities. No sensation is given by a current of less than 10 milliamperes except in cases of auditory hyper-excitability.

The auditory reaction is not infrequently produced in the opposite ear. This phenomenon is known as the *paradoxical reaction*, and indicates that the ear detecting the sound is the affected ear in the case of a unilateral lesion, and the more seriously affected in a bilateral lesion. This hyper-excitability is met with in otitis media and interna, in hyperæmic affections of the labyrinth, and in all conditions which increase intra-cranial pressure (cerebal tumours, meningitis, etc.). On the other hand, absence of reaction to 20 milliamperes indicates grave changes in the two labyrinths.

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[1] Roques, *Arch. d'électr. méd.*, 1911.

CHAPTER V

VOLTAIC VERTIGO

THE infinite variety of head wounds, and the large percentage of cases involving ear trouble, deafness, and vertigo from fracture or concussion, have led to a study of voltaic vertigo in relation to these different conditions. It was shown by the work of Babinski and his pupils that of all the methods of testing the condition of the internal ear, voltaic vertigo is one of the most sensitive.

This at least is the view of the majority of otologists and neurologists, although some consider the modus operandi of the phenomenon too obscure, and regard other tests, rotatory, caloric, and galvanic nystagmus, as adequate for diagnostic purposes.

Galvanic Nystagmus.—When the ear is stimulated by passing a current of increasing strength between one tragus and the other, a slow simultaneous movement of both eyes towards the positive pole occurs when the strength of the current reaches 2 or 3 milliamperes ; this is followed by small and rapid movements in the opposite direction, *i.e. towards the negative* pole, constituting *fine rotatory nystagmus.* To observe this movement, the patient should be told to look downwards, and the operator should watch closely either the pupil or a vessel in the conjunctiva.

In place of the bipolar method, in which a current is passed from one tragus to the other, the unipolar method may be employed, which consists in applying an indifferent electrode to the back of the neck and the negative electrode to the ear which is being tested.

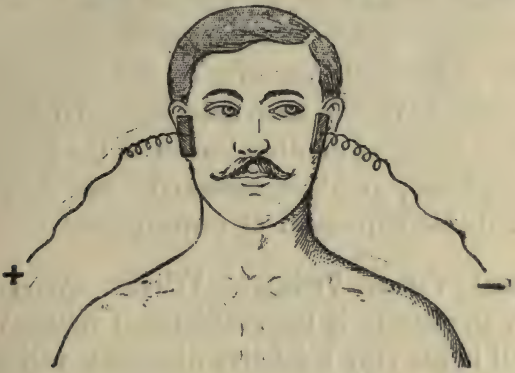

Fig. 43.—Galvanic nystagmus.

A relatively strong current (10 to 20 milliamperes) is required for producing nystagmus by this method, and the vertigo which this will produce renders the procedure decidedly unpleasant for the patient.

Whatever method is used, the movement in the nystagmus is always towards the negative pole in the normal condition.

When nystagmus is produced by a current of less than 2 ma., it may be regarded as indicating a change in the internal ear. This hyper-excitability is the rule in labyrinthine hæmorrhage (Ménière's syndrome), in suppuration in the internal ear, and sometimes in sclerosis. When a current of more than 4 ma. is required, or if nystagmus is not produced by any current which can be tolerated (the limit being about 15 ma.), this hypo-excitability or inexcitability of the ear concerned is generally regarded as indicating grave changes in the posterior labyrinth (partial or total destruction, neuritis of the auditory nerve, etc.).

We are not aware that qualitative changes, as, for instance, abnormal direction of the movements of nystagmus, have ever been described. We have seen, however, a case of gunshot wound of the head, which was trephined, in which nystagmus was replaced by a remarkable movement of convergence.[1]

Normal Voltaic Vertigo. — Voltaic nystagmus is merely an element in the syndrome of voltaic vertigo; in itself it has only a relative diagnostic value, and it may even be absent in the normal state.

In a healthy subject, when two testing electrodes are applied immediately in front of the ear and a current of gradually increasing strength is passed, a series of *subjective* and *objective* phenomena is produced. With 1 to 2 ma., the patient first experiences a sensation of giddiness; with from 2 to 5 ma., other subjective phenomena appear—a metallic taste, phosphenes, ear-noises, marked vertigo, malaise, and nausea. At the same time objective phenomena occur, viz. nystagmus and *inclination of the head towards*

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[1] A. Zimmern, *Soc. neurol.*, April 1917.

the positive pole, sometimes so pronounced that the patient may fall backwards. A current of 1 to 2 ma. will usually cause this movement; 5 ma. will always produce it in a normal person. But with a current of slowly increasing strength, the resulting slow and gradual movement may escape observation; in such a case, if the circuit is broken, a sudden further inclination of the head will usually occur, followed by return to the erect position.

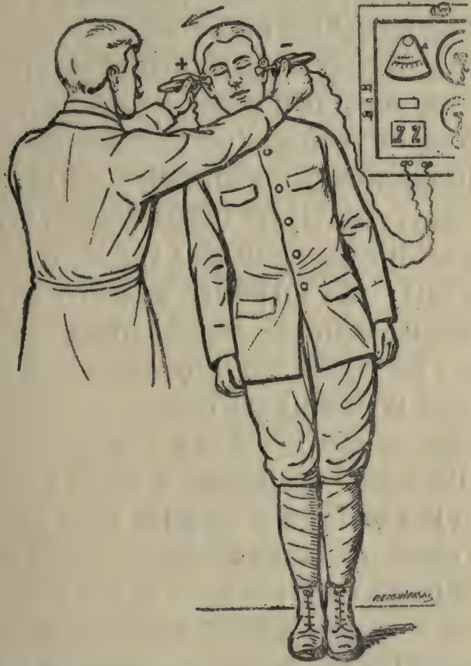

Fig. 44.—Normal voltaic vertigo. The patient is leaning towards the + pole.

When the electrodes are applied asymmetrically and at different levels—if, for instance, the positive electrode is placed above the usual point and the negative below it, below the lobe of the ear and behind the mandible—the movement produced is a *rotation* of the head on its vertical axis, towards the positive pole.

In young subjects the sensation of giddiness is particularly marked, and quite a weak current (1 to 2 ma.) is sufficient to produce it.

Erb, in experiments on himself, made the following observations: One side of the body seems to have lost its weight, with the result that the body falls on the opposite side; the subjective sensation of unsteadiness is greater than the actual movement produced; if the eyes are closed, the body seems to have moved farther than is actually the case.

According to Escat, the objective reaction, that is to say, the lateral inclination, is much more evident if the subject is standing with the heels together and the eyes closed, *i.e.* in the attitude assumed for obtaining Rhomberg's sign (fig. 44). In this position there is not only a movement of the head, but an inclination of the whole body towards the positive pole.[1]

Otologists frequently employ the unipolar method, placing the positive electrode on the back of the neck and the negative over the ear, as was described in connection with nystagmus; with this arrangement a stronger current is required for producing vertigo, and its effects are sometimes less easily tolerated than if the bipolar method is employed.

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[1] The various vestibular tests produce reactional movements which may spread to all parts of the body simultaneously. Each reactional movement is followed by a counter-reactional movement in opposite direction. In voltaic vertigo, the reaction is easily produced, and the counter-reaction is immediate (Babinski).

It is essential in carrying out these tests to repeat them a number of times in succession, when the reaction being constant, the conclusion will be beyond doubt. At the same time care should be taken not to repeat the stimulus at very short intervals, since the effect of the current diminishes, and movement is therefore no longer produced.

Voltaic Angular Deviation.—If a healthy individual be asked to close his eyes after making a note of a certain direction, and then to take six steps forward in this direction, followed by six steps back in the opposite direction, it will be found that he does not deviate appreciably from his original line of direction. This is the test of spontaneous angular deviation suggested by Babinski and G. A. Weill.[1]

In a subject whose mechanism of equilibrium is affected, the deviation is such that if he is stopped after several turns backwards and forwards, he will find himself facing in an opposite direction from that in which he started, or at least considerably to the right or left of it.

If this walking test be applied to a healthy subject simultaneously with Barany's thermal test, deviation occurs in a direction opposite to that of the nystagmus produced (Buys). With cold water, there is deviation towards the side of the ear in which it has been applied.

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[1] Babinski and G. A. Weill, *Soc. biol.*, April 26th, 1913.

More accurate results are obtained with bitemporal electrical stimulation during the walking test, the electrodes being kept in position over the tragus by elastic bands, and connected by wandering leads to the galvanic apparatus. In a normal individual, a current of 1 to 1½ ma. causes inclination towards the side of the anode; the direction taken alters towards the positive pole, a deviation of 10° to 15° being produced in each forward or backward course. Consequently, when the ground has been covered forwards and backwards four times the total deviation will be 190°. This is voltaic angular deviation.[1] The actual course pursued assumes the form of a star.[2]

If the test be repeated immediately after breaking the circuit, deviation towards the opposite side is produced; this is known as voltaic counter-deviation.

With a current of more than 2 ma., the shoulder falls towards the positive pole, the gait is asymmetrical, and phenomena of interference with equilibrium may even appear as drunken, staggering gait and reflex movements aimed at restoring equilibrum (Cestan).

The Normal Vestibular Reactions.—In brief, the following reactions are given by healthy individuals with no lesion of the ear:

1. Nystagmus, the movement being towards the negative pole.

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[1] Babinski and G. A. Weill, *Soc. biol.*, July 19th, 1913.

[2] Cestan, Descomps, and Sauvage, *Soc. Méd. des Hop.*, May 1916.

2. Inclination towards the positive pole, whatever its position, whether over one ear or the other, with a current of 2 to 5 ma.

3. Voltaic angular deviation towards the positive pole.

These normal reactions are given, not only by healthy individuals, but by individuals suffering from *psychical deafness.* But a deaf person with normal reactions should not be regarded on this account as suffering from functional deafness or as a malingerer. Only anomalous reactions possess a definite diagnostic significance.

It is generally recognised that abnormalities of voltaic vertigo are the result of *vestibular lesions* ; deafness from an organic cause will therefore be unaccompanied by abnormal vertigo unless the lesion spreads to the vestibule.

Abnormal Vestibular Reactions.—Although the relation between modifications of voltaic vertigo in the pathological state, and the pathological changes concerned is still imperfectly understood, clinical experience has firmly established the following conclusions:

Two kinds of modification must be considered—quantitative changes in the strength of current required for producing inclination, and changes in the direction of the inclination.

(*a*) *Quantitative Modifications :*

There is said to be *hyper-excitability* when an extremely disagreeable sensation of vertigo is rapidly produced, and continues after stimulation ceases; when inclination is caused by a current of 1 to 3 ma.; or again, when the extent of the movement is exaggerated. In certain cases, very weak currents are liable to produce an unsteadiness of the head, which moves in all directions—forwards, backwards, and laterally; this condition has been called cephalic nystagmus.

This hyper-excitability occurs in acute or chronic suppurative otitis, more particularly during the development of acute labyrinthitis (Hautant).

Hypo-excitability, sometimes known as *resistance to vertigo*, is characterised by the fact that the subjective sensation of vertigo can only be produced with difficulty, and that 10 to 15 ma. are required for producing inclination.

Hypo-excitability may be unilateral, inclination on one side being produced by a current of 2 to 5 ma., and on the other only by 10 ma. or more.

In these cases of hypo-excitability, rotation, angular deviation, and nystagmus are equally affected, all three requiring abnormally strong currents. Nystagmus may even be absent.

When the objective reactions of vertigo are entirely absent, there is said to be *inexcitability* or *absolute resistance* to vertigo.

Hypo-excitability occurs in chronic changes in the labyrinth (grave suppurative labyrinthitis), in chronic otitis media with involvement of the labyrinth, in sclerosis of the internal ear, and lastly in head injuries (fracture, concussion, etc.). It is usually present in intracranial lesions which cause an increase in the pressure of the cerebro-spinal fluid.

Inexcitability would appear to indicate destructive changes in both labyrinths, or at least a very serious bilateral lesion (fractures of the base).

(*b*) *Changes in Direction of the Inclination*:

Anomalies in the direction of the inclination usually coexist with one of the foregoing quantitative changes. Hyper-excitability or hypo-excitability, however, do not necessarily imply such an anomaly: the inclination may be normal in direction, even in severe labyrinthine lesions. Hautant found no changes in the direction of the inclination in cases of suppurative labyrinthitis, or after trephining to expose the labyrinth.

Anomalies of inclination are of three types:

1. There may be simply *predominance of amplitude*. The positive pole still determines the direction of the inclination, but the extent of the movement is always greater on one side; this indicates a change in the ear of that side if the affection is unilateral, or a severer lesion on that side if the affection is bilateral.

2. The inclination is *invariably towards the same side*. Whatever the position of the positive pole, the head inclines towards the side on which the labyrinth is affected, or affected in greater degree, according to the formula stated by Babinski in 1903.

This rule is not, however, invariable. There may be inclination only towards the unaffected side, as in complete unilateral destruction of the vestibular mechanism; or there may be inclination towards the affected side when the positive pole is applied to that side, and a rapid backward movement when it is applied to the unaffected side.

3. In some cases inclination is *absolutely irregular*, sometimes to one side, sometimes to the other; in other cases the movement is always backward. These phenomena are often accompanied by great diminution in excitability, and appear to indicate bilateral lesions.

The Vestibular Reactions in Head Injuries, Concussion, and After Trephining.—A. *Voltaic Vertigo.*—Testing voltaic vertigo in cases of head injury has shown that the reactions are commonly abnormal.

It is not essential that the petrous portion of the temporal bone should be involved: abnormalities of vertigo may follow fracture of any part of the skull, or even simple concussion.

Injuries of the skull often leave the patient with sensations of giddiness of various kinds, disorders of equilibrium, and disorders of hearing. These are usually accompanied by changes in the objective reactions, consisting of modifications of excitability, diminished or absent nystagmus, and abnormal direction of inclination. It is not exceptional to find profound changes in voltaic vertigo where the clinical manifestations are slight (slight vertigo, and reduced acuteness of hearing).

These changes indicate that the subjective symptoms are of organic origin, and dispose of the idea that the disorder is psychical or simulated (Babinski).

Cestan regarded quantitative changes as an almost invariable feature in head injuries. Hypo-excitability is present in cases where trephining has been performed or a certain amount of bone has been removed, and hyper-excitability in concussion and injuries of the mastoid and occipital.

A number of different changes in the direction of the inclination would appear to be possible, since in cases which Babinski studied after trephining, the normal inclination and rotation were replaced either by unilateral inclination, unilateral rotation, inclination and rotation on one side only, inclination on one side and rotation on the other, circumduction of the head, forward and backward movements of the head, or, lastly, by cephalic nystagmus.

B. *Voltaic Angular Deviation.*—The effect of the galvanic current on forward movement has been studied notably by Cestan, Descomps, Euzière, and Sauvage,[1] who observed changes in the type, direction, and degree of angular deviation.

In place of the star-shaped line of movement, which they regarded as normal, they frequently observed a lateral deviation (curve or zig-zag) with one or both positions of the positive pole.

As with the inclination in vertigo, there may be univocal angular deviation independent of the position of the positive pole.

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[1] Cestan, Descomps, Euzière, and Sauvage, *Soc. Méd. Hop.*, October 27th, 1916.

Lastly, whereas the angle of deviation in a healthy individual is about 90°, this may be increased or reduced in pathological conditions, and is sometimes even greater in one direction than in the other. Equal extent of deviation in both stages of the test (deviation and counter-deviation) is exceptional in disease.

The diagnostic value of these changes is similar to that of abnormal voltaic vertigo. Cestan further asserts that whenever vertigo is abnormal, the deviation produced by an electric current will indicate similar conclusions. It must not be assumed, however, that the results of these two tests will always be analogous in form; to give only a single example, bilateral inclination in vertigo may be accompanied by unilateral angular deviation. These facts do not militate against the value of the method when it is applied, not for the purpose of discovering the exact situation or nature of the lesion, but in order to prove that the subjective symptoms of which the patient complains have some basis in actual organic change.

The Theory of Anomalies in Voltaic Vertigo.—*Nystagmus* is produced by stimulation (rotation, heating, or galvanisation) of one of the vestibules, the vestibular nerve, or of its nuclei in the medulla.

In the vestibule, the reflex appears to originate more particularly in the ampullary region of the semicircular canals. The hair-cells of the crista acustica are affected by movements of the endolymph, and transmit this stimulus to the terminations of the vestibular nerve.

Barany's caloric nystagmus is usually regarded as being produced by an ascending current in the endolymph, its temperature in the external semicircular canal being raised; this effect has even been regarded as explaining galvanic nystagmus in cataphoresis and galvanotropism of the hair-cells (Brunings).

It is difficult, however, to believe that in caloric nystagmus the liquid injected into the external ear will raise the temperature of the internal ear sufficiently to produce convection currents. Further, such currents would hardly affect the walls of the canals, and consequently the hair-cells; in a capillary tube about the size of a pin (*i.e.* of the same diameter as the semicircular canals), filled with liquid of a certain viscosity, movement is almost entirely confined to the centre of the column, and the liquid in contact with the walls of the tube is almost stationary.

The possibility of electrical convection seems equally unlikely, in view of the minute size of the semicircular canals, and consequently the insignificant fraction of the current which traverses them.

On the contrary, it seems much more reasonable to suggest that here, as elsewhere, the galvanic current acts directly by stimulating the vestibular nerve. It has been shown that galvanic nystagmus not only occurs in lesions of the labyrinth, but also in cases in which the nerve endings are affected.

Voltaic vertigo, properly so-called, also originates in stimulation of the posterior labyrinth.

Babinski's experiments on the pigeon, and those of Weill, Barré, and Vincent on the guinea-pig, leave no possibility of doubt on this point. Experimental galvanic stimulation of the labyrinth in the animal produces the phenomena of voltaic vertigo, and destruction of the labyrinth abolishes them.

In the guinea-pig, the rotation normally produced by the action of the positive pole is generally absent after unilateral extirpation of the labyrinth. It can only be produced by applying the positive pole on the opposite side. After bilateral extirpation no movement can be produced.

Extracranial section of the auditory nerve gives similar results. It is therefore evident that the vertigo is not a purely labyrinthine effect, but depends on stimulation of the centripetal fibres (vestibular nerve) originating in the labyrinth.

The mechanism of the reflex itself, the muscles concerned in the movements produced, the relation of these movements to labyrinthine tonus, and the specific action of the positive pole, represent questions of which, as yet, we know very little.

CHAPTER VI

ELECTRO-DIAGNOSIS AND THE MEDICAL BOARDS

THE importance of electrical diagnosis has been recognised by the army medical service, with the result that the report on an electrical examination has been given adequate prominence in Medical Board papers.

Every case of paralysis of any kind or degree should be sent for electrical examination.

When it is remembered that the Medical Board, the Minister, or even Council of State which represents the final court of appeal in awarding pensions, have only medical reports from which to draw their conclusions, it will be recognised that these reports should include, whenever possible, unquestionable scientific data, which will verify the clinical evidence and assist the decisions of experts.

Camus has shown how much can be gained from physiological means of examination (the dynamo-ergograph and measurement of the vaso motor and psycho-motor reactions): of these procedures, electrical examination is second to none in importance.

There will be no question of a paralysis when a recent electrical examination confirms its existence.

Further, although a clinical examination will determine the extent of functional impairment, the electrical reactions will often indicate the prognosis, an important factor in estimating the extent of a disability, and particularly in deciding whether a man is to be discharged from the army, discharged temporarily, or transferred to an auxiliary branch.

It was hoped by some that electrical methods would afford objective evidence of the functional power of nerves and muscles, and that the formulæ—complete RD, partial RD, etc.—would be of value as indicating definite degrees of disability. Attempts have also been made to class the various stages of RD in degrees, according to the state of faradic and galvanic excitability, the polar formula, the nature of the contraction, and the longitudinal reaction, each of these signs to indicate a further stage in degeneration. Such a classification as this would obviously provide a scale of degrees of severity which would enable any medical officer, apart from special qualifications, to form conclusions with a minimum of difficulty. But this system is far from corresponding to clinical reality; it may rightly be applied in cases of recent injury, but it is wide of the mark in long-standing paralyses, such as appear before Medical Boards. Such cases may have improved, or complete functional recovery may have taken place, but at the same time, as members of Medical Boards have very rightly been reminded by the *Guide-barème des Invalidités*, voluntary movement is recovered before regressive changes in the electrical reactions have begun. The results of electrical examination may therefore be identical in two cases, in one of which function has to a great extent been recovered, while in the other nothing beyond muscular tone has been regained. If the electrical reactions were taken as sole guide, the extent of the disability would be assessed as equal in the two cases.

Larat has proposed simplifying the terminology by abolishing the expression "partial RD," a condition which he himself regards as indicating an early and rapid improvement. We are not of the opinion that the term should be abolished, since partial RD may occur, either as a preliminary stage to complete RD, or, on the other hand, during the regressive period, where it reflects the traces of the lesion that remain.

Actually, however, whatever term may be employed to describe a group of abnormal electrical reactions, it cannot adequately inform a Medical Board of the exact condition of affairs. It is only by interpreting the electrical reactions and correlating them with clinical observations that a conclusion can rightly be drawn.

What information can the Medical Board obtain from an electrical examination?

The presence of abnormal electrical reactions differentiates at once between organic paralyses and simulated, hysterical, or functional paralyses (Babinski type). In the majority of cases, when the case appears before the Medical Board, an electrical examination merely confirms the clinical diagnosis.[1] We have already mentioned that it is common for a functional paralysis to be associated with an organic lesion: in such cases, an electrical examination will differentiate between what is due to hysteria and what to organic change.

An electrical examination for the purposes of a Medical Board often reveals an unsuspected lesion. If, for instance, the Board is faced with an ankylosis of the knee following an arthrotomy for articular fracture, and this is found to be accompanied by interference with the movements of the foot, electrical examination will often reveal an unrecognised lesion of the external popliteal nerve, the foot-drop having previously been attributed to the prolonged immobilisation or to muscular atrophy. We have mentioned elsewhere that paralysis of the ulnar may pass unrecognised apart from an electrical examination. Similarly, in cases which have been in hospital for some months and are in process of being boarded out of the army, we have frequently discovered grave nerve lesions with complete RD, which had previously been unsuspected; in some cases a bone lesion was the principal injury (in articular fracture of the wrist with ankylosis, for instance, paralysis of the muscles of the thenar eminence may remain unnoticed), in others, it may only have been observed that one nerve is paralysed, whereas another is equally affected. It seems hardly necessary to emphasise the importance, from the point of view of the man's own interests, of carrying out this additional examination.

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[1] During the first two years of the war, we frequently found in the medical history sheets of cases sent to us for electrical examination, observations implying an organic lesion of a peripheral nerve, whereas the reactions were found to be absolutely normal. Proceeding with a clinical examination, we generally found that the case was one of functional paralysis. Duco and Blum describe numerous cases in which a further examination ordered by the Board led to alteration of the original diagnosis of an organic lesion. Some months ago, specialists for conducting neurological examinations were attached to all Medical Boards, in districts where there was no neurological centre. Unrecognised hysterical paralyses are now very rarely to be met.

In organic paralyses of peripheral nerves, an electrical examination will determine the actual condition of the lesion. It is not sufficient to carry out a rapid examination and then to say that RD is or is not present; it is also necessary to determine the stage of the degeneration and to interpret these data, to find whether the electrical reactions suggest a lesion capable of repair, or a permanent lesion, since the decision as to the degree of disability and future of the man concerned (discharge, temporary discharge, or transfer to an auxiliary branch), which depend on the results of this examination and on the clinical evidence, will be decidedly different in each case.

Hypo-excitability may be found to be the only anomaly in the electrical reactions; in some cases it is the only remaining trace of an RD in process of regression, in others it is entirely independent of RD.

It occurs in simple muscular atrophies, but also in certain nerve lesions, the clinical signs of which suggest a grave condition. Simple hypo-excitability is usually a favourable sign.

Partial RD also suggests a favourable prognosis in the majority of cases; it may be present either in slight paralyses or during nerve regeneration.

Complete RD indicates a grave nerve lesion. Its manner of evolution being variable, its significance in prognosis depends on whether that evolution appears to be progressive or regressive. The nerve, as well as the muscles, should be tested, since it is in the former that the first signs of regressive change appear. Longitudinal hyper-excitability is also an important sign, but is not in itself a sufficient reason for concluding that the paralysis is incurable: in toxic neuritis, for instance, longitudinal hyper-excitability is common, but recovery usually takes place. In the vast majority of cases, reliable conclusions can only be based on repeated examinations, or on sufficiently explicit reports of previous electrical examinations.

Absolute inexcitability must logically be regarded as indication that the lesion is incapable of repair. It is certainly an extremely grave sign, but it must be remembered that after suitable physiotherapeutic treatment, or an operation (suture or liberation of the nerve), inexcitability may persist for a long time before the appearance even of the earlier effects of successful treatment. It is therefore advisable in some cases to postpone the decision when the condition is suspected of being incurable. When the lesion dates from more than a year previously, when muscular atrophy is marked, and accompanied by marked trophic disorder, it is almost impossible that function can even partially be recovered.

Compensation for injuries received, either on active or during home service, takes one of two forms, a pension or a gratuity. By the terms of the Act of 1831, which is still in force, a pension is only awarded when the disability is both severe and incurable. This is a pension for life, and even though it may have been granted owing to an error in diagnosis or prognosis, the State has no alternative, and it must continue to be paid; hence the importance of avoiding haste in deciding that a nerve lesion is incurable.

A gratuity is granted when the extent of functional impairment is at least 10 per cent.; the condition may be curable within a variable length of time, even such that recovery is comparatively rapid. According to the nature of the case, the man is either discharged permanently, in which case the gratuity awarded will be paid every two years, or he is discharged temporarily, which means that he may be called up again for active service twelve months later, for which period he receives the gratuity awarded. Thirdly, a man recommended for a gratuity may be transferred to auxiliary service if his disability is not so serious as to be incompatible with such service, in accordance with the stated instructions as to physical fitness for military service.

The testing of voltaic vertigo, together with the examination of the cerebro-spinal fluid in cases of head injury (trephining or concussion), became compulsory, following a meeting of the Société de Neurologie in April 1916. This was a fortunate decision. since abnormal vertigo demonstrates the objective nature of the disorders (vertigo and headache) of which the patient complains. Abnormalities in vertigo should be taken into account in the decisions of a Medical Board, but, as with the muscular reactions, they can only be a single factor in that decision. Lortat-Jacob requested that no head case with abnormal vertigo should return to active service; Laignel-Lavastine will never send a man back to his depôt until his vertigo is normal. As a matter of fact, it is impossible to state a definite rule.

In short, although electro-diagnosis cannot determine the precise nature of a nerve lesion, it is of great value to the Medical Board as an objective sign of an organic lesion and as a factor in deciding the prognosis. It is therefore absolutely essential that the examination should be exactly and carefully carried out.

In every case a report should be written, dealing not merely with the elementary reactions obtained, but giving clear and concise conclusions. We have already pointed out the faults of arbitrary classifications ; nothing so clearly illustrates the ever-recurring difficulty in forming conclusions as the apparent discrepancies by which we are sometimes momentarily nonplussed. The decision of the electrologist is a matter of judgment, for all cases have their individual character.

DOCUMENTS

I

39 C 1/7. *March* 15*th*, 1916.

Organisation of medico-legal procedure in cases recommended for pensions and permanent discharge

You are instructed to draw up what is known as the *Dossier médical* of the man in question; that is to say, the combined reports which will enable specialists such as the members of the consulting commission to ascertain the cause, the exact nature, the origin, the degree of gravity, and the condition of incurability or non-incurability of diseases and disabilities.

This medico-legal dossier contains the reports of duly qualified specialists (ophthalmologists, oto-rhinologists, neurologists, etc.), the results of electrical examinations, radiographic examinations, etc.; in fact, all data essential for judging of the gravity and the incurability or non-incurability of the disabilities concerned.

II

GUIDE-BARÈME DES INVALIDITÉS, 1915

Paralysis of Nerves

Every case of paralysis involves a study of the impairment of movement and sensation, the condition of the reflexes, and vaso-motor, trophic, and electrical disorders.

The latter in particular are of the greatest importance, from the point of view both of the gravity of the lesion and of the prognosis, and consequently of estimating the degree of disability.

It is of particular importance to determine whether RD is present. Its presence definitely indicates a lesion of the peripheral motor neurone.

RD may be complete or partial.

Complete RD appears in cases of complete division of the nerve, and in many cases of compression. It corresponds to *complete paralysis*.

Partial RD occurs in compression and irritation of the peripheral motor neurone. It corresponds to *incomplete paralysis*.

When complete RD is observed, the condition is *grave*, and *often incurable*.

It should not be concluded prematurely, however, that the paralysis is incurable: this conclusion is only permissible when prolonged and vigorous treatment has been without effect.

Nerves, like all other tissues, when they have been injured, have a natural tendency to undergo repair, and in this process the axis-cylinder is the first element to regenerate. Now this axis-cylinder is capable of transmitting an electrical stimulus, and in accordance with the law stated by Duchenne (of Boulogne), the power of voluntary movement reappears earlier than electrical excitability.

If, on the other hand, the lesion is in process of becoming incurable, the nerve remains permanently inexcitable, and in the case of the muscle, hypo-excitability is succeeded by inexcitability.

If RD is partial, the prognosis is more favourable; the condition is curable, and if recovery is not certain, it is at least extremely probable.

III

348 C 1/7 CCM. *December 5th*, 1916.

Medical examination of cases of head wounds from the point of view of fitness for further service

Le Sous-Secrétaire d'Etat du Service de Santé militaire to MM. les Directeurs du Service de Santés des Régions.

The question of the fitness for service of men who have received head wounds has been the subject of numerous discussions. At the meeting of members of the Société de Neurologie de Paris and representatives from military neurological centres, the problem was examined, and it was decided that from facts hitherto observed and interpreted the following conclusions could be drawn.

In the first place, head injuries involving grave organic lesions of the nervous system, such as hemiplegia, monoplegia, loss of speech, very extensive losses of bone, etc., obviously incompatible with any form of military service, must be excluded.

Apart from these cases, it is necessary in every case of head injury to examine for a number of other disorders, the presence of which is important from the point of view of the prognosis : headache, phosphenes, dizziness ; labyrinthine, visual, and mental disorders ; abnormalities in voltaic vertigo ; the emotional, sympathetic, circulatory, and secretory reactions ; and lastly, excess of albumen in the cerebro-spinal fluid, etc.

As regards the course to be adopted in cases in which these disorders have been present, or are still present, it appears difficult and undesirable to formulate arbitrary instructions ; account should always be taken of the individual nature of the case.

It may, however, be stated as a principle that men who have received head wounds should not return to the front, apart from altogether exceptional cases which have been submitted to prolonged observation.

The procedure indicated is usually temporary discharge, or transfer to an auxiliary branch of the service.

JUSTIN GODART.

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